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Vol. 15 No. 3 JULY, 1961

BRITISH JOURNAL OF PREVENTIVE & SOCIAL MEDICINE

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CONTENTS

PAGE

Anencephalus in Scotland. R. G. RECORD 93

The Relation between Smoking Habits and Physique, Respiratory Symptoms, Ventilatory Function, and Radiological Pneumoconiosis amongst Coal Workers at Three Scottish Collieries. J. R. ASHFORD, S. BROWN, D. P. DUFFIELD, C. S. SMITH, and J. W. J. FAY 106

Multiple Sclerosis in British Commonwealth Countries in the Southern Hemisphere. E. D. ACHESON 118

Incidence of Gastric and Duodenal Ulcer in the Royal Navy in 1955. J. H. BARON and R. VAUGHAN JONES 126

Prevalence of the Commoner Ear, Nose and Throat Conditions in the Adult Rural Population of Great Britain. R. HINCHCLIFFE 128

Society for Social Medicine 141

LONDON
BRITISH MEDICAL ASSOCIATION
TAVISTOCK SQUARE, W.C.1

YEARLY SUBSCRIPTION (4 NUMBERS) £3 U.S.A. $10.00 SINGLE NUMBER 17s. 6d.

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Fluhmann, C. F. (1936). *Amer. J. Obstet. Gynec.*, **31**, 573.
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Brit. J. prev. soc. Med. (1961), **15**, 93

ANENCEPHALUS IN SCOTLAND

BY

R. G. RECORD

From the Department of Social Medicine, University of Birmingham

In the search for causes of congenital malformations many methods have been used. Animal experiments have revealed a large number of teratogenic agents and have provided some understanding of the mechanism of maldevelopment. Embryological studies have indicated the probable stage of development when various malformations are initiated. Examination of pedigrees has shown a genetic basis for some of the less common or less lethal human malformations. New cytological techniques have contributed substantially to our understanding of mongolism and a few infrequent abnormalities. But so far these researches have thrown little light on the causal factors of the majority of human malformations.

It is perhaps surprising that population research methods have not been more widely used, particularly in view of the fact that the most notable advance in the field of human teratology — the incrimination of rubella—was achieved by these means. A possible reason is the difficulty of organizing an inquiry on a scale large enough to provide adequate numbers for analysis. It might be expected that national statistics would meet this need, but unfortunately they rarely supply data on stillbirths in sufficient detail for examination. Scotland is the only country which publishes adequate records on stillbirths classified according to cause and distinguishing between the commoner malformations—anencephalus, hydrocephalus, spina bifida, and cardio-vascular abnormalities. A series restricted to stillbirths is hardly adequate for study of the last three malformations, many of which survive birth, but it is well suited for an investigation of anencephalus which is rarely compatible with a post-natal existence even for a few hours. (Of the 349 anencephalics born in Scotland in 1958, all but nineteen were stillborn.) Another characteristic which makes anencephalus a useful subject for investigation on a national scale is its unmistakable appearance, even to an inexpert observer. A third reason is that its relatively high frequency enables a large series to be assembled from the data now available. Indeed the present importance of anencephalus as a cause of stillbirth gives some urgency to the investigation of its aetiology. In 1938–41 the Scottish stillbirth rate was 41·3 per 1,000 total births; by 1956–58 it had been reduced to 23·5, but the anencephalic stillbirth rate had risen in the same period from 2·39 to 2·99. The contribution of anencephalus to the stillbirth rate therefore increased from 5·8 to 12·7 per cent.

An extensive analysis of Scottish data has been reported by Edwards (1958). He confirmed the findings of an earlier survey in Birmingham (Record and McKeown, 1949) that the incidence of anencephalus is higher for first births than for second births and for foetuses born in the late autumn and winter months than for those born in the summer. Edwards found a more pronounced association with maternal age than was shown by the Birmingham data, but could not exclude the possibility that this might be the result of an association with parity. His findings of a marked association with social class was consistent with Aberdeen experience (Anderson, Baird, and Thomson, 1958) but differed from the Birmingham data which showed no variation of incidence with social circumstances. He considered that the secular variation in incidence was due to changing incidence among first births and among winter births. The point is an important one, for if confirmed it would help to narrow the search for aetiological agents. For the same reason an examination of the relation of seasonal variation to parity and social class would be useful

The Registrar General for Scotland does not publish data which permit a study of this detail, but he consented to supply a break-down of the Scottish statistics on anencephalic stillbirths, so that a fuller examination is now possible.

Method

The new data here analysed relate to legitimate anencephalic stillbirths in Scotland for the years 1949–58 classified according to:

(1) Age of mother and number of previous children;
(2) Month of registration of birth and number of previous children;
(3) Month of registration of birth and social class.

Other details of anencephalic stillbirths and data relating to all births (live and stillborn) during the period were obtained from the annual reports of the Registrar General for Scotland. In some parts of the analysis, numbers have been augmented by using data for earlier years (1939–48) from the same source. Incidence in the two periods is compared in Table I.

Results

Since this analysis is based both on new data which relate only to legitimate births and on published statistics which in some instances include illegitimate births, it may be useful to begin by investigating the influence of illegitimacy on incidence. This will be followed by consideration of the influence of maternal age and parity and of seasonal and secular variations in incidence.

Illegitimacy

During the period 1939–58 there were 274 illegitimate anencephalic stillbirths—2·45 per 1,000 total births (Table I). This is 0·14 (approximately 1 standard error) below the population value of 2·59, but since only about 5 per cent. of all births are illegitimate the difference in anencephalic rates between legitimate births and all births is negligible and there seems to be little need to discriminate between the two sets of data.

The slightly low anencephalic rate among illegitimate births is, however, of some intrinsic interest. Standardization for maternal age raises the difference from 0·14 to 0·20, and it seems likely that further standardization for parity and social class (not permitted by the available data) would increase the difference to a significant level.

Part of the difference can be accounted for by the fact that in some areas where the anencephalic rate is low the illegitimacy rate is high. For example, the fifteen counties north of a line joining the Firths of Clyde and Tay experienced in the years 1950–58 an anencephalic rate of 2·0 per 1,000 and an illegitimacy rate of 5.8 per cent., the corresponding rates for the remainder of Scotland being 3·0 per 1,000 and 4·3 per cent. From data for the same period for each of the 33 counties, the coefficient of correlation between the two rates (each value being weighted according to the number of births) was found to be −0·4. Areas of low anencephalic incidence therefore contribute an undue proportion of births to the illegitimate series. It seems unlikely, however, that this accounts completely for the low anencephalic rate among illegitimate births, which has been observed elsewhere (Record and McKeown, 1949). Another explanation, plausible but so far not supported by evidence, is that attempts to procure abortion (presumably a considerable hazard for illicit pregnancies) are more likely to succeed when the foetus is anencephalic than when it is normal.

Maternal Age and Parity

Since 1939, the Registrar General has published data on live births and stillbirths related to maternal age, distinguishing between legitimate and illegitimate births, but the distinction is not maintained when stillbirths are classified according to cause and to maternal age. The published data on parity are based on legitimate births, but the Tables for 1939–45 differ in form from those for 1946–58 so that a satisfactory combination of the two periods is not possible. The published data on age and parity have the further disadvantage that they do not permit a separate

TABLE I
INCIDENCE OF ANENCEPHALIC STILLBIRTHS

Period	Incidence	Legitimate	Illegitimate	Total
1939-48	Total No. of Births	917,135	66,673	983,808
	No. of Anencephalic Stillbirths	2,266	152	2,418
	Incidence per 1,000 Total Births	2·47	2·28	2·46
1949–58	Total No. of Births	916,968	45,037	962,005
	No. of Anencephalic Stillbirths	2,499	122	2,621
	Incidence per 1,000 Total Births	2·73	2·71	2·72
Total	Total No. of Births	1,834,103	111,710	1,945,813
	No. of Anencephalic Stillbirths	4,765	274	5,039
	Incidence per 1,000 Total Births	2·60	2·45	2·59

examination of these rather highly correlated variables. For these reasons, attention will here be restricted to the new data supplied by the Registrar General relating to legitimate births for the years 1949–58.

Crude results are shown in Fig. 1. Incidence is high in first pregnancies; it is lowest in second pregnancies, and then rises steadily with advancing parity. Incidence by maternal age shows a similar but more symmetrical pattern, being low in women aged 25–29 and high in the youngest and oldest groups. Since age and parity are positively correlated, one of these patterns could easily be a reflection of the other. The separate effects of the two variables are examined in Table II. In some of the age/parity cells, numbers of births are inevitably small; it has therefore been necessary to combine the two highest age groups of Fig. 1, and also the highest parity groups. The columns of the Table show that the parity effect demonstrated in the Figure is followed consistently by both the older groups. The three younger groups naturally provide less information about the higher parities, but no major inconsistencies are revealed. All five groups show low rates for second pregnancies. Perusal of the rows of the Table suggests that the U-shaped maternal age pattern shown in Fig. 1 is made up of several components, first pregnancies contributing mainly to the descending left arm of the U, and third and later pregnancies to the ascending right arm. The low rates in second pregnancies appear not to vary with maternal age.

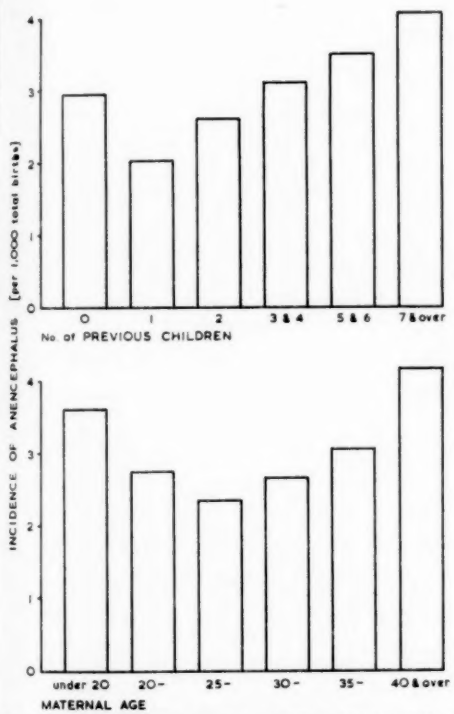

FIG. 1.—Anencephalic stillbirth rates (per 1,000 total births), 1949–58, by maternal age and parity.

TABLE II

ANENCEPHALIC STILLBIRTH RATES (PER 1,000 TOTAL BIRTHS) ACCORDING TO MATERNAL AGE AND PARITY (LEGITIMATE BIRTHS, 1949–58)

No. of Previous Children	Maternal Age (yrs)				
	Under 20	20–	25–	30–	35 and Over
0	3·9	3·1	2·5	2·5	2·7
1	(1·9)	2·2	2·0	2·1	2·0
2	—	1·9	2·4	2·8	3·4
3 and 4	—	(2·8)	2·8	3·0	3·7
5 and Over	—	—	(2·3)	3·4	4·2

Figures in brackets are based on less than 10,000 births

In Scotland there is a very marked social gradient in the incidence of anencephalus which is four times higher in Social Class V than in Social Class I (Edwards, 1958). The social classes also show pronounced differences in reproductive habits, Social Classes I and II being well represented among the lower birth ranks at the higher ages, whereas the higher birth ranks at the lower ages come mainly from Classes IV and V. Social class variation in incidence might therefore account for a positive correlation of incidence with parity and would tend to mask an increase of incidence with maternal age. It follows that standardization for social class would tend to reduce the apparent effect on incidence of high parity and increase the effect of advanced maternal age. In fact, although standardization brings about both these changes (Table III, overleaf), its effect is not so marked that the results outlined in the previous paragraph need re-appraisal.

It is concluded that the incidence of anencephalus varies both with parity and with age, being high among first-born, especially when the mother is very young, and among the higher birth ranks, especially when the mother is old.

TABLE III

ANENCEPHALIC STILLBIRTH RATES STANDARDIZED FOR SOCIAL CLASS

No. of Previous Children	Maternal Age (yrs)				
	Under 20	20–	25–	30–	35 and Over
0	3·6	3·1	2·6	2·9	3·0
1	—	2·1	2·0	2·3	2·1
2	—	1·7	2·3	2·9	3·5
3 and 4	—	—	2·7	3·0	3·8
5 and Over	—	—	—	3·0	4·0

SEASONAL VARIATION

In a previous report (McKeown and Record, 1951), the incidence of anencephalus in Birmingham and in Scotland was shown to be higher in autumn and winter than in spring and summer, the fourth quarter of the year (October–December) having the highest rate and the second quarter (April–June) the lowest. In that investigation, division of the year into orthodox quarters was necessary because of the form of the Birmingham data, but it does not reveal the full extent of the seasonal variation. In fact, it is the least efficient of the three possible ways of dividing the year into trimesters in order to examine fluctuations in incidence; the maximum range and variance are obtained if the first trimester starts in February (Table IV). This is the basis for the grouping of the months used in the analysis which follows.

TABLE IV

INCIDENCE OF ANENCEPHALUS IN VARIOUS TRIMESTERS OF THE YEAR LEGITIMATE AND ILLEGITIMATE BIRTHS, 1949–58

Trimester	Incidence (per 1,000 total births)	Range	Variance
January–March ..	2·82	0·73	0·08
April–June ..	2·42		
July–September ..	2·53		
October–December ..	3·15		
February–April ..	2·57	1·02	0·14
May–July ..	2·30		
August–October ..	2·74		
November–January ..	3·32		
March–May	2·39	0·79	0·10
June–August	2·47		
September–November ..	2·89		
December–February ..	3·18		

Parity.—The numbers of anencephalics, grouped according to parity, who were born in each trimester are shown in Table V. For each parity the relative incidence according to season was calculated by dividing the proportion of anencephalics born in each trimester by the proportion of all births born in the same period. For example, for the first parity,

TABLE V

NUMBERS OF LEGITIMATE ANENCEPHALIC STILLBIRTHS, ACCORDING TO SEASON AND PARITY, 1949-58

No. of Previous Children	Trimester of Birth				Total
	February–April	May–July	August–October	November–January	
0	221	205	251	295	972
1	132	120	128	152	532
2 and 3	153	135	157	193	638
4 and Over	86	84	81	101	352
Not Stated	3	2	—	—	5
Total ..	595	546	617	741	2,499
Total Births	245,740	248,641	233,563	234,061	962,005

the relative incidence in the period February–April was

$$\frac{221}{972} \Big/ \frac{245740}{962005} = 0{\cdot}89.$$

The four parity groups show a consistent pattern (Fig. 2). In all of them incidence is highest in the period November–January and lowest in May–July. The seasonal fluctuation is least marked for the highest parities (four or more previous children), but this may be due merely to the smaller size of this group.

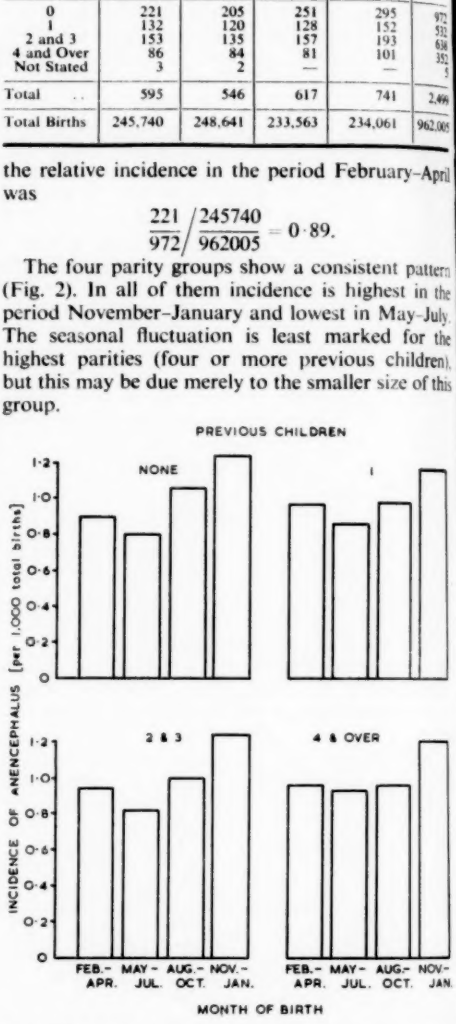

FIG. 2.—Relative incidence of anencephalus by season according to parity, 1949–58.

Unfortunately, there are no published data on the seasonal distribution of all births classified according

to parity and it has been necessary, therefore, to assume that the seasonal pattern of human reproduction does not vary with parity. The validity of this assumption was explored by examining data relating to all births in Birmingham where the necessary information is collected. Births occurring in 1950 were distributed in the same parity and season groups as before and for each parity group the percentage of births in each trimester was calculated (Table VI). There is very little difference between the four parity groups, all showing the lowest proportion of births in November–January and the next lowest in August–October. The only inconsistency is among first parities where most births occur in May–July whereas the maxima of the other three groups are in February–April. These differences are so trivial when compared with the marked seasonal fluctuation shown in Fig. 2 that it seems unnecessary to attempt to apply corrections.

TABLE VI

PERCENTAGE DISTRIBUTION BY SEASON OF BIRTHS (LIVE AND STILL), ACCORDING TO PARITY BIRMINGHAM, 1950

No. of Previous Children	No. of Births	Trimester of Birth				Total
		February–April	May–July	August–October	November–January	
0	7,142	25·2	26·1	24·7	24·1	100
1	5,395	26·2	25·7	24·1	24·0	100
2 and 3	4,500	26·8	25·7	24·6	22·9	100
4 and Over	1,879	26·7	26·4	23·7	23·2	100
Total . .	18,916	26·1	25·9	24·4	23·7	100

Social Class.—Results of a similar analysis in respect of social class are shown in Table VII and Fig. 3.

TABLE VII

NUMBERS OF LEGITIMATE ANENCEPHALIC STILLBIRTHS, ACCORDING TO SEASON AND SOCIAL CLASS, 1949–58

Social Class	Trimester of Birth				Total
	February–April	May–July	August–October	November–January	
I and II	48	50	42	73	213
III	313	308	359	409	1,389
IV	112	98	115	136	461
V	122	90	101	123	436
Total . .	595	546	617	741	2,499

Social Classes I and II have been combined because of their relatively small numbers of anencephalics. Classes III, IV, and V have seasonal patterns which closely resemble each other; in all, the highest rates occur in November–January and the lowest in May–July, and each class shows about the same

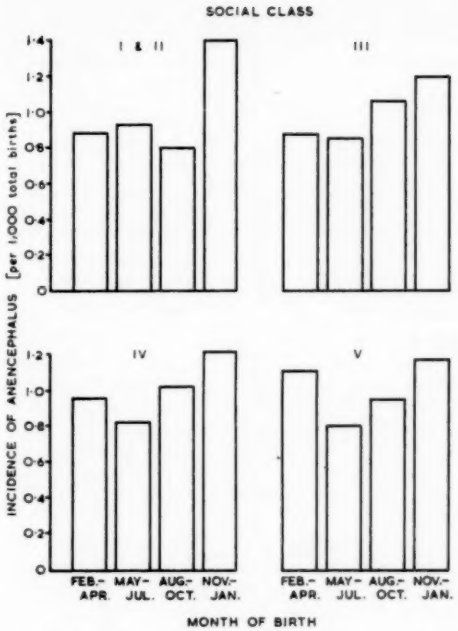

FIG. 3.—Relative incidence of anencephalus by season according to social class, 1949–58.

range, the highest rate being 1·4 to 1·5 times the lowest. Social Classes I and II also show the highest incidence in November–January, but the rate is almost constant in the other three periods. It is possible that the relatively small size of this group has caused the low May–July incidence shown by the other classes to be obscured by random variation.

There is no information on social class variation in the seasonal distribution of the total population of births, and it has been assumed that there are no class differences in this respect. To justify this assumption, data on Birmingham births have again been examined. These data are not quite so helpful as before, since they permit identification of only three social classes. Social Classes I and II of the Registrar General are approximately equivalent to Group A in the Birmingham series, III and IV to B, and V to C. The seasonal distributions of births thus classified are very similar (Table VIII, overleaf).

It is concluded that the seasonal trends of anencephalus shown in Fig. 3 approximate reasonably closely to the true values.

Reasons for Seasonal Variations.—There can be no doubt that the incidence of anencephalus fluctuates

TABLE VIII

PERCENTAGE DISTRIBUTION BY SEASON OF BIRTHS (LIVE AND STILL), ACCORDING TO SOCIAL CLASS BIRMINGHAM, 1951–2

Social Class	No. of Births	Trimester of Birth				Total
		February–April	May–July	August–October	November–January	
A	4,895	25·9	26·7	24·3	23·0	100
B	26,825	25·7	25·8	24·4	24·0	100
C	4,929	25·3	26·4	24·1	24·2	100
Total ..	36,649	25·7	26·0	24·4	23·9	100

widely with season and it seems that this variation extends to all classes and to all parities—even to the more privileged classes and to second pregnancies, where the general incidence is low. A satisfactory exploration of the reasons for the seasonal variation would involve consideration of the many aspects of the environment which vary with season. This is too wide a subject for full investigation at present and attention will here be restricted to certain climatic phenomena and some of the more common infectious diseases.

Before attempting to correlate the seasonal variation of possible influences with monthly anencephalic rates, it must be recognized that, because the duration of gestation of anencephalic foetuses is so variable, the date of birth is only a rough guide to the date when the causal agent acted upon the embryo. Also, since gestation is usually shorter than normal and since there is a seasonal variation in the total number of births, some error is introduced when the anencephalics born in any month are related to the total births in that month; a substantial proportion should be related to the births of the following month and some to the month after that. (The error is, of course, less when a 3-monthly grouping is adopted as in the first part of this section.) Examination of Birmingham data indicates that, in round figures, only one-sixth of anencephalics are born in the month expected (counting from the date of the last menstrual period), one-half being born one month early and one-third 2 months early.

In order to calculate monthly incidence figures, it is necessary, therefore, to advance one-half of the anencephalics born in a given month to the following month, and one-third to the month after that. The time of operation of the causal factor, supposing that it acts when the embryo is about 3 weeks old, would then be 8 months before the adjusted month of birth. In using this method, it is necessary, because the number of days in a month is not constant, to convert the monthly figures to daily numbers before carrying out the adjustment. Results of analysis of all the available Scottish data (1939–58) are shown in Table IX. These suggest that, unless the factor responsible for the seasonal incidence has a delayed action affecting primarily the mother rather than the embryo, it operates most strongly from March to July and least strongly from September to December.

The adjusted anencephalic rates of Table IX, related to the month when the influence was presumed to operate, have been charted in Fig. 4 (opposite) against the mean monthly figures for mean air temperature, daily hours of daylight, and hours of sunshine in West Scotland. (This area was chosen because it contains the greater proportion of the population.) The scales have been adjusted to make the ranges approximately the same; comparison should be based, therefore, on the inflexions of the curves and the position of the modes rather than on the proximity of the lines. The daylight and sunshine curves are, of course, very similar; both fit the anencephalic variation more closely than the tempera-

TABLE IX

MONTHLY ANENCEPHALIC STILLBIRTH RATES—SCOTLAND, 1939–58

Month of Birth	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
Total No. of Days ..	620	565	620	600	620	600	620	620	600	620	600	620
Total Births in Month ..	165,618	150,929	171,629	169,420	177,221	164,552	167,058	157,821	151,554	164,762	147,200	158,049
Total Births per Day ..	267·13	267·13	276·82	282·37	285·84	274·25	269·45	254·55	252·59	265·74	245·33	254·92
Anencephalic Stillbirths in Month	509	429	412	413	369	386	342	401	379	455	436	508
Anencephalic Stillbirths per Day	0·8210	0·7593	0·6645	0·6883	0·5952	0·6433	0·5516	0·6468	0·6317	0·7339	0·7267	0·8194
Anencephalic Stillbirths per Day corrected for Short Gestation*	0·7887	0·8102	0·7641	0·7000	0·6649	0·6343	0·6119	0·5980	0·6126	0·6537	0·6987	0·7446
Monthly Corrected Anencephalic Stillbirth Rate	2·95	3·03	2·76	2·48	2·33	2·31	2·27	2·35	2·43	2·46	2·85	2·92
Month of Operation of Influence	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.

*Corrected numbers are obtained by advancing half the anencephalics 1 month and one-third 2 months.

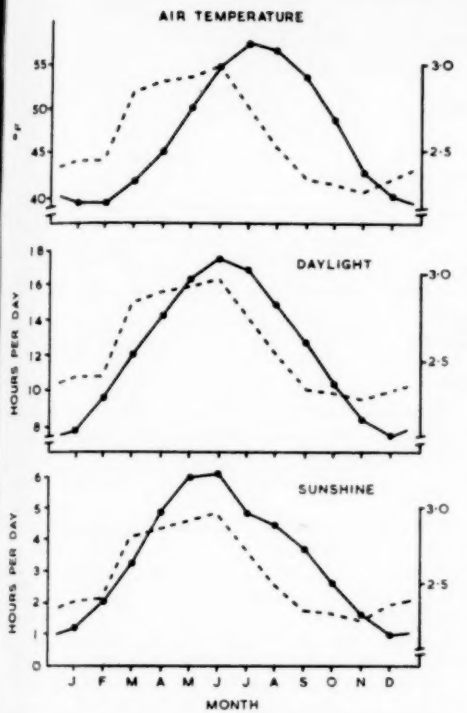

FIG. 4.—Comparison of mean air temperatures, daylight hours, and sunshine hours (continuous lines) with anencephalic stillbirth rates 8 months later (broken line).

ture curve does, but although the positions of the modes coincide the agreement is not perfect.

There is much less agreement, however, with some of the commoner infectious diseases (Fig. 5, overleaf). Choice of the diseases investigated depended on the availability of weekly notifications and, since the Registrar General does not publish these figures, data for Glasgow (published in the *Glasgow Medical Journal*) have been used. There seems little objection to using figures for Glasgow, which is the centre of the largest conurbation in Scotland with a seasonal distribution of disease presumably not very different from that of other heavily populated areas which are not far away. The chief limitation of the data is that not all infectious diseases are notifiable, and only a few of those that are occur with a frequency which makes an investigation of the seasonal trend useful. Scarlet fever, whooping cough, chicken pox, and poliomyelitis show no correspondence with the anencephalic curve. Measles fits more closely and rubella is even closer, but this is presumably a chance association because the teratogenic effects of rubella have been thoroughly investigated and there is no evidence that it causes anencephalus.

Influenza deserves special consideration in view of the evidence provided by Coffey and Jessop (1959) of its association with anencephalus. It is not a notifiable disease, but a reliable guide to the prevalence of virus A, which is the type most likely to be concerned, is provided by mortality data. These show a seasonal pattern quite unlike the seasonal variation of anencephalus (Fig. 6, overleaf). The implication of this observation is supported by an examination of secular variations of the two conditions. Between 1938 and 1958 there were seven epidemic winters (arbitrarily distinguished by more than 500 deaths from influenza in the period October to March); only two (in 1939–40 and 1957–58) were followed the next May to October by an anencephalic incidence above the normal value. The fact that the second of these epidemics was the one investigated by Coffey and Jessop raises the possibility that the causal virus, known to differ antigenically from its predecessors, also differed in its teratogenic potentialities. Two contemporary American studies, however, showed no obvious association with anencephalus (Wilson, Heins, Imagawa, and Adams, 1959; Walker and McKee, 1959). Doll, Hill, and Sakula (1960), in London, also obtained negative results, but concluded, after examining Scottish statistics, that Asian influenza in early pregnancy "can increase the risks of anencephaly, but that the extent of the hazard is normally small".

Further study of the Scottish data suggests that the risk is negligible. If influenza has any teratogenic action, the duration of the illness and of the period during which the embryo is at a stage when it is susceptible to the development of anencephalus suggest that the danger to the embryo lasts for at least 3 or 4 days. In Scotland, therefore, where there are about 8,000 births each month, at least 1,000 embryos are at risk at any time. The 1957 epidemic was largely restricted to the months of October and November, during which about 25 per cent. of women of reproductive age were attacked. There were therefore about 500 embryos exposed to maternal influenza at the relevant time; three-quarters of those which developed anencephalus would be expected to be born in the following May and June. The actual number of anencephalic stillbirths registered in these months (62) was greater than the numbers in 1957 and 1959 when there was no preceding autumn epidemic (36 and 54 respectively) but

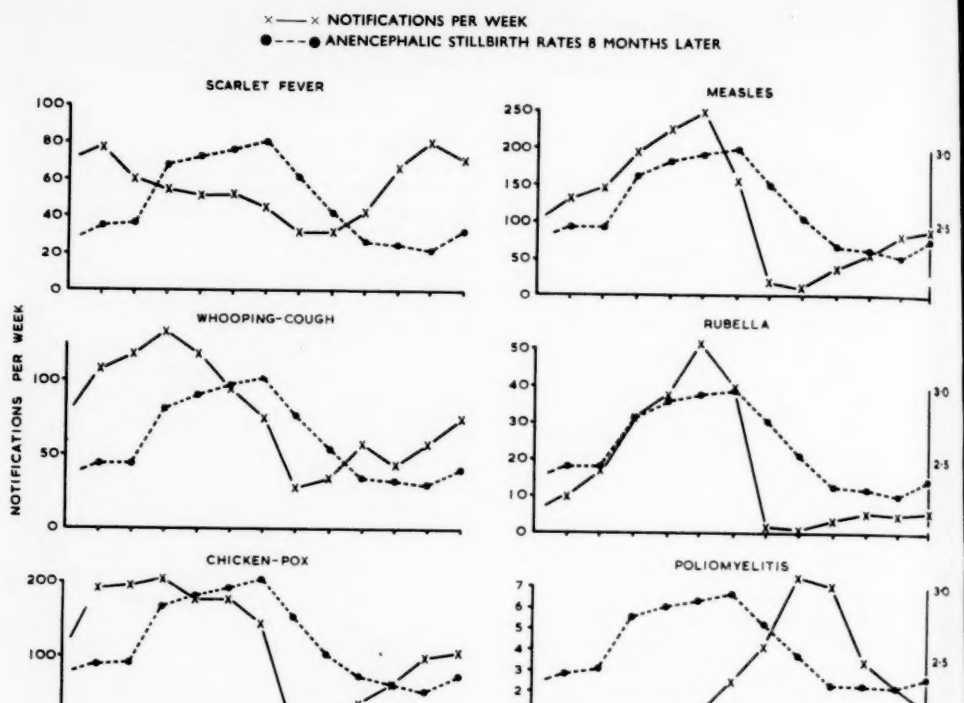

FIG. 5.—Comparison of monthly frequency of infectious diseases in Glasgow in 1945–54 (continuous lines) with adjusted anencephalic stillbirth rates 8 months later (broken line).

was very much less than the number which would have occurred if influenza were an important influence.

A second epidemic due to the same virus strain in February and March, 1959, provided a further opportunity to study its effect. The number of anencephalic stillbirths born in the following September and October was 42; the previous year, when there was no prevalence of influenza in late winter, 58 anencephalic births were recorded in these months.

These results support the view that influenza cannot be regarded as a specific cause of anencephalus. If there is any association, it is possible that this occurs, as Doll and others (1960) suggest, only when other circumstances are suitable.

Secular Variation

Annual anencephalic rates in Scotland from 1939 to 1956 were examined by Edwards (1958), who considered that much of the fluctuation from year to year was due to changes in incidence among first births and among winter births, rates for multiparae and for summer births showing little more than random variation. It is worth noting, however, that annual rates may be misleading because anencephalic foetuses are usually born prematurely; a substantial proportion of those born towards the end of a year would, if normal, have been born in the following year and should therefore be related to the births of that year. No error arises, of course, if the annual

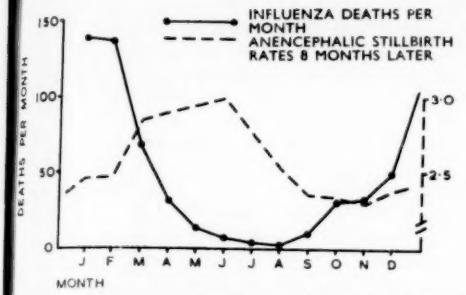

FIG. 6.—Comparison of monthly frequency of deaths from influenza in Scotland (continuous line) with adjusted anencephalic stillbirth rates 8 months later (broken line).

number of total births is constant, but if, as in 1945, the number of births in one year is smaller than the number in the following year, the anencephalus rate is overstated. The error can be minimized by grouping consecutive years as has been done in a later part of this analysis.

In attempting to explain changes in the annual rates in terms of variation in the environment, it is more useful to know incidence according to the year when the malformation was initiated than to the year of birth. These rates can be readily calculated if it is assumed, as was done in the examination of seasonal variation, that the malformation is laid down about 3 weeks after conception (approximately 8 calendar months before term). Normal infants born in the period September–December therefore pass through this stage in the same year as the year of birth, and infants born in January–August pass through it in the previous year. The same rule can be applied to anencephalic foetuses if their month of birth is first adjusted as in the previous section to take account of their shorter period of gestation.

Rates derived in this way are shown in Fig. 7.

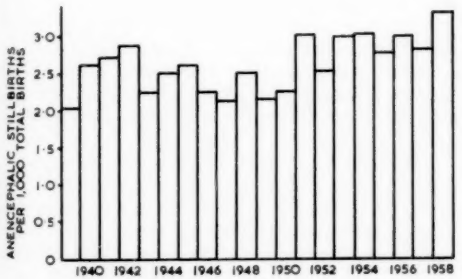

FIG. 7.—Incidence of anencephalic stillbirths according to year in which the malformation was initiated.

Minor fluctuations from year to year are of doubtful significance (the standard errors are in the range 0·15–0·18 per 1,000) and only the broader trends merit attention. It appears that incidence increased during the early war years and then settled to a low level which persisted until 1950; since then rates have been high, especially in 1958. At first sight this pattern suggests a direct relationship between anencephalic rates and food intake. (Although food rationing was introduced at an early stage of the war it became more severe and persisted well into the post-war period, fat consumption for example reaching its lowest level in 1947.) It has not been possible, however, to find any close correlation between anencephalic rates and the national intake of the major nutrients. Similar attempts to correlate incidence with the prevalence of the notifiable infectious diseases considered in the previous section were equally unrewarding. Nor is there any relationship between rates and the more obvious meteorological phenomena.

A full investigation of the many environmental factors subject to annual variation which might be concerned in the causation of anencephalus would be very laborious and is hardly justifiable unless the number of possible agents can be narrowed. At present the most useful line of inquiry seems to be an investigation of secular trend in relation to those variables which have been shown to have some influence on incidence—season, maternal age and parity, and social class.

The available data permit examination of the secular influence of season of birth and maternal age for the whole period 1939 to 1958 (Table X, overleaf). To reduce random error due to the small numbers which occur in some cells when the series is divided according to these variables, a 5-year grouping has been adopted. The anencephalic rate was highest in 1954–58 and lowest in 1944–48. Each of the four seasons of birth also showed their highest rates in 1954–58, but only two (May–July and November–January) were consistent with the general pattern in having their lowest rates in 1944–48. These two seasons also provided the greatest range in the quinquennial rates. It seems that the seasons with the lowest and the highest rates (May–July and November–January respectively) show the greatest secular variation.

Dividing the series according to maternal age gives a rather different pattern. All groups had their highest rates in 1954–58 and all but the youngest had their lowest rates in 1944–48, but the amount of secular variation was small for the group with the lowest incidence (25–29 years), and the group with the

TABLE X

SECULAR VARIATION IN ANENCEPHALIC RATES (PER 1,000 TOTAL BIRTHS) ACCORDING TO SEASON OF BIRTH AND MATERNAL AGE

Quinquennium		1939–43	1944–48	1949–53	1954–58	Ratio of Highest to Lowest
Trimester of Birth	February–April	2·51	2·54	2·33	2·80	1·20
	May–July	2·18	1·88	1·99	2·60	1·38
	August–October	2·44	2·50	2·54	2·94	1·20
	November–January	3·05	2·68	3·21	3·42	1·28
Maternal Age	Under 25	2·20	2·35	2·59	3·07	1·40
	25–29	2·22	2·19	2·23	2·50	1·14
	30–34	2·71	2·31	2·36	2·97	1·29
	35 and Over	3·40	2·89	3·03	3·58	1·24
All Births		2·53	2·39	2·51	2·94	1·23

highest incidence (35 and over) showed less variation than the youngest group.

Incidence according to maternal parity could be examined only for the years after 1945 because in the earlier years birth ranks of stillbirths and live-births were estimated in different ways. The first quinquennium of Table X has therefore been omitted and a 3-year period (1946–48) has been substituted for the second quinquennium. Incidence was low in this period; it increased in 1949–53 and was highest in 1954–58 (Table XI). The first three birth ranks were consistent with this pattern, but later-born children showed an irregular trend. Secular variation in incidence appeared to be most marked for second pregnancies (in which rates are lowest). This contrasts sharply with the secular pattern according to maternal age where the group with the lowest rate showed the least variation. Further exploration of this point was attempted by comparing incidences in the last two quinquennia in respect of both age and parity, but numbers in some cells were too small to establish any consistent pattern.

TABLE XI

SECULAR VARIATION IN ANENCEPHALIC RATES ACCORDING TO MATERNAL PARITY (LEGITIMATE BIRTHS)

No. of Previous Children	Period			Ratio of Highest to Lowest
	1946–48	1949–53	1954–58	
0	2·31	2·84	3·04	1·32
1	1·52	1·77	2·34	1·54
2	2·04	2·29	2·96	1·45
3 and 4	3·10	2·69	3·55	1·32
5 and Over	3·64	3·82	3·69	1·05
Total	2·25	2·50	2·94	1·31

Examination of secular variation according to social class was possible only for the period 1949–58, and in order to maintain adequate numbers a 5-year grouping was adopted and Classes I and II were amalgamated. The results show that the increased incidence in 1954–58 was experienced by all social classes (Table XII).

TABLE XII

SECULAR VARIATION IN ANENCEPHALIC RATES ACCORDING TO SOCIAL CLASS (LEGITIMATE BIRTHS)

Social Class	Quinquennium		Ratio $\frac{b}{a}$
	1949–53 (*a*)	1954–58 (*b*)	
I and II	1·35	1·76	1·30
III	2·57	2·91	1·13
IV	2·73	3·32	1·22
V	3·25	3·93	1·21
Total	2·50	2·94	1·18

DISCUSSION

This investigation confirms previous observations that the incidence of anencephalus varies with maternal age and parity, social class, and season. It is now clear that each of these variables has an independent effect; association with maternal age, for example, cannot be explained by the influence of parity, and the composite effect of age and parity is not due to the social class variation. Furthermore, association with one variable does not nullify the effect of another; the relationship with season of birth, for example, appears to be manifested by all parity groups and by all social classes, and a consistent secular variation is shown by most of the subgroups derived by dividing the series according to maternal age, parity, social class, or season of birth.

Before considering the implications of these findings, it is necessary to examine the possibility that they might be the result of bias due to incomplete ascertainment.

Anencephalus is so easily recognized that it seems unlikely to be missed or confused and there is little doubt that the only cases not recorded are those born alive, and those born dead before the 28th week of pregnancy. The proportion born alive appears to be small. It was 5 per cent. in Birmingham in 1940–47; the same figure was recorded for Scotland in 1958 (the only year for which this information is available).

This proportion is too small to bias results seriously even if survival at birth was dependent to a large extent on one of the variables under consideration.

The abortional loss is certainly greater and requires closer scrutiny. Some idea of its magnitude may be obtained from a survey of all pregnancies in Belfast in 1957 (Stevenson, Dudgeon, and McClure, 1959). In 84 cases of abortion in which the foetus was available for examination, five were anencephalic. If this frequency is representative of the whole series of 1,127 abortions (which occurred in 9,526 pregnancies) it can be estimated that the loss of anencephalics is seven per 1,000 pregnancies. This is higher than the anencephalic rate in pregnancies which reach a viable stage (4·6 per 1,000 total births). A substantial proportion of cases are missed, therefore, when attention is restricted to pregnancies which have reached a viable stage (28 weeks), and we must consider whether the association with the variables examined in the present study might be due to selective elimination of malformed foetuses at an early stage of pregnancy.

For example, the variation in incidence by maternal age could be accounted for, even if the initial incidence were unrelated to age, if women aged 25–29 had a greater tendency to abort anencephalic foetuses than younger or older women. Although there is no direct evidence on this point, there are grounds for believing that this supposition is highly unlikely. The data published by Stevenson and others (1959) provide estimates of the relative incidence of abortions according to age. Comparison with the Scottish anencephalic statistics shows a remarkable similarity in trend when both series are standardized to remove variation due to parity (Fig. 8). (Standardization is necessary because the incidence of anencephalus but not of abortion is raised in first pregnancies.) This strongly suggests that loss by abortion cannot account for the observed variation of anencephalic incidence with age; on the contrary it would probably tend to obscure the true extent of the variation.

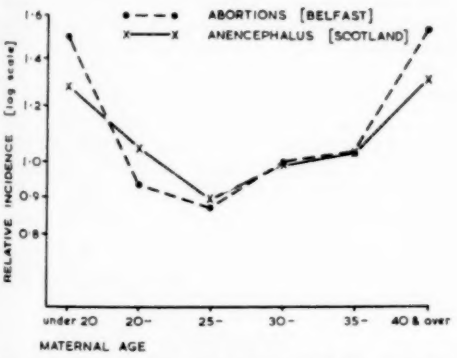

FIG. 8.—Relative incidence of anencephalus and of abortion according to maternal age.

In the same way, again using Stevenson's data, it can be shown that the parity trend of abortions is inconsistent with the view that the parity pattern of anencephalus can be explained by differential abortion loss.

This evidence, of course, does not dispose of the possibility that the age and parity pattern may be different for abortions which occur very early, perhaps even before the fact of pregnancy is established. Such abortions are probably frequent and there is evidence (though derived only from women with genital disease) that a high proportion of such embryos are abnormal (Hertig, Rock, Adams, and Menkin, 1959). All that can be said at present is that, if there is a differential loss of embryos at this stage, it must show a very unusual pattern if it can account for any of the variations in anencephalic incidence so far reported.

Since it is unlikely that the association between anencephalus and the variables examined in this paper can be explained in terms of differential survival of affected foetuses, the relationships here demonstrated are presumably of aetiological significance. It has been shown that these variables are independent of each other in their influence and are to some extent additive in their effect. For example, in Scotland, the risk of an anencephalic stillbirth is always considerable for a woman in poor social circumstances, but the risk is increased if conception takes place in early summer.

It seems likely, therefore, that anencephalus may be due to a number of causes. This conclusion is consistent with laboratory experience; Giroud (1960) listed sixteen agents which can produce anencephalus in experimental animals. It is also quite possible that anencephalus is not an entity and that different types may originate in different ways. Some cases may arise by failure of fusion of the anterior part of the neural folds and sometimes might be associated with spina bifida. These would, perhaps, show the epidemiological features of spina bifida rather than of anencephalus (for example, they might show no seasonal variation in incidence), and spina bifida may be as likely as anencephalus among subsequent members of the sibship. In other cases the neural folds may fuse normally and the anencephalic state may arise later by destruction of nervous tissue. If this were so, the causal agent would act a little later than was supposed in the analysis of seasonal variation, and the dotted curves of Figs 4, 5, and 6 should be shifted slightly to the right. The possibility that anencephalus could arise in this way should stimulate search for a neurotropic virus with a teratogenic

action. Investigation of the viruses of poliomyelitis and mumps by Manson, Logan and Loy (1960) and of ECHO 9 by Peterson and Glicklich (1960) have yielded negative results, but there are many more known neurotropic viruses (and perhaps many not yet discovered) which await study.

The present analysis provides some evidence consistent with the hypothesis that some cases of anencephalus may be due to an infective agent. Incidence was high among:

(1) Young mothers (whose immunity might be supposed to be lower than that of older persons);

(2) Mothers of high parity (the presence of many children in the home would increase the risk of infection);

(3) Mothers in poor social circumstances (which would favour transmission of infection).

The seasonal and secular variations could also be compatible with an infective cause. Findings which do not support an infective cause are the increased incidence among primiparae and at high maternal ages. These associations suggest hormonal disturbance as a possible factor, but other hypotheses worth considering are the less efficient uterine circulation of primiparae and the greater risk of faulty gametogenesis in older women.

Although the malformation has been initiated in experimental animals by excess of vitamin A, and by deficiency of pantothenic acid, vitamin E, and folic acid, epidemiological studies provide little support for the view that anencephalus in man is due to nutritional factors. It is difficult to account in nutritional terms both for the social class gradient and for the low incidence during the years when rationing was most severe, or for the very different experience immediately after the war in parts of Germany, where the rate reached an unprecedented level, and in Scotland, where it was low, and in the last year of the war in Holland where, in spite of severe famine, no obvious increase in anencephalus occurred.

It may be remarked that no mention has been made in this paper of the possible influence of inheritance. The reason is not that hereditary causes are regarded as totally unimportant, but that the nature of the present investigation precluded any examination of a genetic basis for this malformation. It is worth noting, however, that there are several twin studies which provide strong evidence against a simple genetic explanation. Furthermore, the lethal nature of the malformation is not easily reconciled with the perpetuation of a gene or genes, which, in order to account for this condition, would be quite common, at least in some countries. Perhaps the genetic constitution of the embryo is less important than the maternal genotype, which may influence the environment of the early embryo so as to produce conditions favourable for the development of the abnormality. It would be unwise to disregard the possible importance of genetic factors so long as ethnic variations in incidence cannot be adequately explained in environmental terms. For this reason, population studies in other countries could make an important contribution to our understanding of this malformation.

Summary

Unpublished data on anencephalic stillbirths in the years 1949–58 provided by the Registrar General for Scotland, and less detailed statistics from his Annual Reports for the years 1939–48, were examined in relation to information on all births contained in the Annual Reports covering these periods.

The anencephalic rate was slightly lower than normal among illegitimate births. Variations of incidence with age and with parity showed U-shaped trends, being lowest for mothers aged 25–29 years and for second pregnancies. The effects of these two variables were independent of each other and of social class influences.

Incidence was highest in infants born in the period November–January, and this was shown by all parity groups and all social classes. Adjustment of rates to the probable time when the malformation was initiated indicated that the seasonal influence was strongest from March to July. Comparison of this pattern with that of the more frequent notifiable infectious diseases showed little resemblance; daylight and sunshine hours gave a closer correspondence.

Secular variation of the whole series was compared with that of sub-groups obtained by dividing according to season of birth, maternal age, parity, and social class. A high incidence in the last quinquennium was consistently shown by all these sub-groups.

It is concluded that several factors are probably involved in the aetiology of anencephalus, but that much of the present evidence would be consistent with an infective agent as the cause. There is less evidence to support a nutritional hypothesis.

I am most grateful to the Registrar General for Scotland for providing detailed statistics of anencephalic stillbirths for the years 1949–58.

References

Anderson, W. J. R., Baird, D., and Thomson, A. M. (1958). *Lancet*, **1**, 1304.

Coffey, V. P., and Jessop, W. J. E. (1959). *Ibid.*, **2**, 935.
Doll, R., Hill, A. Bradford, and Sakula, J. (1960). *Brit. J. prev. soc. Med.*, **14**, 167.
Edwards, J. H. (1958). *Ibid.*, **12**, 115.
Giroud, A. (1960). *In* "Ciba Foundation Symposium on Congenital Malformations", p. 199. Churchill, London.
Hertig, A. T., Rock, J., Adams, E. C., and Menkin, M. C. (1959). *Pediatrics*, **23**, 202.
Manson, M. M., Logan, W. P. D., and Loy, R. M. (1960). Ministry of Health: Reports on Public Health and Medical Subjects, No. 101. H.M.S.O., London.
McKeown, T., and Record, R. G. (1951). *Lancet*, **1**, 192.
Peterson, J. C., and Glicklich, L. (1960). *Amer. J. Dis. Child.*, **100**, 779.
Record, R. G., and McKeown, T. (1949). *Brit. J. soc. Med.*, **3**, 183.
Stevenson, A. C., Dudgeon, M. Y., and McClure, H. I. (1959). *Ann. hum. Genet.*, **23**, 395.
Walker, W. M., and McKee, A. P. (1959). *Obstet. and Gynec.*, **13**, 394.
Wilson, M. G., Heins, H. L., Imagawa, D. T., and Adams, J. M. (1959). *J. Amer. med. Ass.*, **171**, 638.

Brit. J. prev. soc. Med. (1961), **15**, 106

THE RELATION BETWEEN SMOKING HABITS AND PHYSIQUE, RESPIRATORY SYMPTOMS, VENTILATORY FUNCTION, AND RADIOLOGICAL PNEUMOCONIOSIS AMONGST COAL WORKERS AT THREE SCOTTISH COLLIERIES

BY

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Smoking has for some time been strongly suspected of being a major aetiological factor in chronic bronchitis. Confirmatory evidence has accrued from many epidemiological studies involving widely different populations. Abbott, Hopkins, Van Fleit, and Robinson (1953) found that, in 294 hospital patients with emphysema and cough, tobacco smoking could be incriminated as an aetiological factor in about two-thirds of the cases, and Palmer (1954) confirmed these findings, again on a hospital population.

The use of hospital populations in epidemiological studies is open to the danger of selection, with consequent doubts about the general applicability of the results, but the work of Abbott and Palmer and their collaborators cited above has been confirmed in studies of representative samples of the general population. Clifton (1956), in a study of rural dwellers and of workers in heavy industry in Sheffield, found smoking to be an important factor in the prevalence of chronic bronchitis in these two populations. Ogilvie and Newell (1957), in their Newcastle survey, confirmed the association between smoking and chronic bronchitis, but were unable to relate the prevalence of the disease to the amount of tobacco smoked. Brown, McKeown, and Whitfield (1957), in a study of 1,062 men aged 60–69 years, and Higgins, Oldham, Cochrane, and Gilson (1956), in their Leigh survey, agreed with these findings.

Less information is available about the effects of smoking on ventilatory function. Franklin (1958) found that heavy smokers recorded lower flow rates in maximal forced expirations than did light smokers and non-smokers, this happening frequently in the absence of any pulmonary abnormality. Eich, Gilbert, and Auchincloss (1957), investigating experimentally the effects of cigarette smoking, found, in emphysematous patients, that there was an increase in airways resistance after smoking one cigarette, but Bickerman and Barach (1954) found no significant effect after their subjects had smoked three cigarettes. Higgins (1959), in a study of 734 men aged 25 to 64 years randomly selected from urban and rural populations, recorded a higher indirect maximum breathing capacity (M.B.C.) for non-smokers than for smokers, but found "no significant downward trend with increasing tobacco consumption among the smokers". Fletcher, Elmes, Fairbairn, and Wood (1959) came to the same conclusion about a group of London postmen, aged 40 to 59 years. However, Higgins, in a study with other workers of a population of mixed occupations in Staveley, Derbyshire, found that "the heavier smokers recorded a lower mean M.B.C. than the lighter" (Higgins, Cochrane Gilson, and Wood, 1959).

As part of the National Coal Board's Pneumoconiosis Field Research (Fay, 1957; Fay and Rae, 1959; Rogan, Ashford, Chapman, Duffield, Fay, and Rae, 1961), periodic medical surveys, including the measurement of ventilatory function and physique and the assessment of radiological pneumoconiosis, respiratory symptoms, and smoking habits, are carried out on the underground and surface workers at a sample of 25 collieries distributed throughout the main British coalfields. The data collected on these surveys provide a convenient opportunity to examine the relation between smoking habits and these other factors in complete working populations, and this paper is concerned with the results obtained at the first three collieries in Scotland at which the necessary measurements have been made.

Data Available

The three collieries under consideration are situated respectively in Ayrshire, Lanarkshire, and the Lothians. At each survey more than 90 per cent. of the underground and surface workers on the colliery books were examined and information is available for a total of 4,014 men.

The medical examination can be divided into the following four main components:

(*i*) The taking of a $14'' \times 14''$ chest X-ray film which was subsequently read in accordance with the I.L.O. classification (I.L.O., 1953).

(*ii*) The forced expiratory volume measured in litres in the first second (F.E.V. 1 sec.), using the Gaensler apparatus (Gaensler, 1951), as modified by the Medical Research Council Pneumoconiosis Research Unit (Carpenter, Cochrane, Gilson, and Higgins, 1956). Four sets of readings were recorded, of which the first was discarded to take account of the "learning" effect (Ashford, Forwell, and Routledge, 1960).

(*iii*) The measurement of sitting height, standing height and weight.

(*iv*) The application of a short respiratory symptoms questionnaire, including questions about smoking habits (Appendix).

At all three collieries the data were collected by the same (No. 1) X-ray Unit, under Dr. S. Rae.

Each man presenting himself for examination was asked the following questions about his smoking habits:

(*a*) "Do you smoke?"

(If the answer to (*a*) was "Yes")

(*b*) "How many cigarettes do you smoke each day?"

(*c*) "How much pipe tobacco do you smoke each week?"

(If the answer to (*a*) was "No")

(*d*) "Have you ever smoked as much as ten cigarettes a day for 10 years?"

It will be noted that, for the purpose of replying to question (*d*), "having ever smoked" was interpreted as having smoked at least ten cigarettes per day (or the equivalent in pipe tobacco) for as long as 10 years or the equivalent. This definition was deliberately chosen to be more rigorous than that employed by Doll and Hill (1950), who regard "one cigarette per day for 1 year" as being a sufficient "qualifying" period. However, it has since been found in special inquiries amongst the working populations at three collieries (Rae, 1960) that in practice this difference in definition has only a small effect on the proportion of men classed as "non-smoker". Out of a total of some 4,500 men, there was a change in the smoking classification (from "non-smoker" to "ex-smoker") of only 1·5 per cent. when the criterion was altered from "ten cigarettes per day for 10 years" to "one cigarette per day for 1 year". In view of this small difference and in order to remove the apparent inconsistency between the definitions of "smoker" and "ex-smoker", the more commonly accepted definition of "one cigarette per day for 1 year" has been adopted for subsequent medical surveys.

It will be noted that, apart from question (*d*), the inquiry was confined to current smoking habits. The difficulty of obtaining an accurate and detailed record of a man's smoking history has been emphasized by Todd and Laws (1958), and it was considered that little further information would be gained by attempting to do so in this inquiry.

The general trends shown by the results obtained at the three collieries were similar and all the available data have therefore been taken together in the subsequent analysis.

Summary of Smoking Habits

According to their answers to questions (*a*) and (*d*) the men examined were classified as "Smokers" ['Yes' to (*a*)], "Ex-smokers" ['No' to (*a*), 'Yes' to (*d*)], or "Non-smokers" ['No' to (*a*) and (*d*)]. Thus the use of the term "non-smoker" in this paper is not strictly consistent with the practice of other authors (*e.g.* Todd, 1957), who apply the term to all men who did not smoke at the time the questionnaire was asked, whatever their past smoking history. According to their answers to questions (*b*) and (*c*), the smokers were further subdivided into "Cigarettes only", "Pipe only", and "Cigarettes and pipe"*. The amount of tobacco consumed has been expressed in terms of "equivalent cigarettes per day", one ounce of pipe tobacco being taken as equivalent to 28 cigarettes.

The smoking habits recorded at the three collieries are summarized in Table I and illustrated by Fig. 1(*a*) (overleaf), where the results have been subdivided in terms of six age groups—Under 21, 21–30, 31–40, 41–50, 51–60, and Over 60 years. Approximately 80 per cent. of the men examined were classed as smokers, 17 per cent. as non-smokers, and 3 per cent. as ex-smokers. Under the more commonly accepted definition of ex-smokers, the proportion of men assigned to this group would probably be increased by about 1·5 per cent., with a corresponding

* At the Lothians colliery questions (*b*) and (*c*) were modified and it is not possible to obtain a breakdown of the smokers in terms of "cigarettes" and "pipe".

decrease in the proportion of non-smokers. The proportion of smokers shows very little variation with age. As might be expected on general grounds, the ex-smokers tend to occur most frequently amongst the older age groups.

TABLE I

SUMMARY OF SMOKING HABITS, BY AGE

Age Group (yrs)	Smokers		Non-smokers		Ex-smokers		Total	
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Under 21	280	73·1	103	26·9	0	0·0	383	100
21–30	555	75·1	182	24·6	2	0·3	739	100
31–40	777	82·7	138	14·7	25	2·7	940	100
41–50	755	83·2	110	12·1	42	4·6	907	100
51–60	610	81·7	102	13·7	35	4·7	747	100
Over 60	237	79·5	42	14·1	19	6·4	298	100
All	3,214	80·1	677	16·9	123	3·1	4,014	100

Table II, which refers only to the Ayrshire and Lanarkshire collieries, omitting the Lothians, shows that some 84 per cent. of the smokers were classed as "cigarettes only", 11 per cent. as "pipe only", and 5 per cent. as "cigarettes and pipe". Fig. 1 (*b*) shows that the proportion of cigarette smokers decreases steadily with age, from 100 per cent. for the Under 21-year age group to less than 50 per cent. for the Over 60-year age group. There is a corresponding increase with age in the proportion of pipe smokers and, to a lesser extent, in the proportion of men who smoke both cigarettes and pipe.

TABLE II

SUB-DIVISION OF SMOKERS, BY SMOKING HABITS AND AGE

(excluding Lothians Colliery)

Age Group (yrs)	Cigarettes (only)		Pipe (only)		Cigarettes and Pipe		Total	
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Under 21	149	100·0	0	0·0	0	0·0	149	100
21–30	295	99·0	2	0·7	1	0·3	298	100
31–40	338	90·6	23	6·2	12	3·2	373	100
41–50	368	82·1	52	11·6	28	6·2	448	100
51–60	292	72·8	75	18·7	34	8·5	401	100
Over 60	62	48·8	50	39·4	15	11·8	127	100
All	1,504	83·7	202	11·2	90	5·0	1.796	100

Details of the average tobacco consumption at the Ayrshire and Lanarkshire collieries are given in Table III. The average amount smoked by the cigarette smokers is approximately fifteen cigarettes per day, compared with the equivalent of just over ten per day for the pipe smokers and 25 per day for those who smoke both cigarettes and pipe.

TABLE III

AVERAGE TOBACCO CONSUMPTION BY SMOKING HABITS AND AGE

(excluding Lothians Colliery)
Average No. of Cigarettes per Day (or Equivalent)

Age Group (yrs)	Cigarettes (only)	Pipe (only)	Cigarettes and Pipe	All Smokers
Under 21	10·9	—	—	10·9
21–30	15·3	5·5	15·5	15·3
31–40	16·4	13·1	22·8	16·4
41–50	16·2	10·9	27·2	16·3
51–60	14·9	10·7	25·5	15·0
Over 60	13·8	9·0	21·8	12·9
All	15·2	10·5	25·0	15·2

Fig. 2 (overleaf) shows that, amongst the younger men, the amount of tobacco consumed tends to increase with increasing age, reaching a maximum of the equivalent of just over sixteen cigarettes per day for the 31 to 40 and 41 to 50-year age groups, and then declining to just less than thirteen cigarettes per day for the Over 60-year age group. A similar trend is noted when each of the types of smoker is considered separately.

These results are, in general, in fairly good agreement with the findings of Todd (1957), who carried out sample survey inquiries amongst the general population based on a similar questionnaire. In comparison with Todd's figures for males in Great Britain in 1956 (see Figs 1 and 2), the proportion of smokers in the three colliery populations is slightly higher for all age groups. The proportion of smokers who smoke both cigarettes and pipe is slightly lower than the national average and the proportion who smoke cigarettes only is slightly higher. In all age groups the amount of tobacco consumed by the smokers is slightly less than Todd's national average figures.

PHYSIQUE

In the course of the medical surveys, measurements have been made of the weight, standing height, and sitting height of each man presenting himself for examination. It has been found that standing height provides no further information about ventilatory function additional to that which is provided by sitting height, and for this reason weight and sitting height only have been considered in the subsequent analysis. The average weight corresponding to each

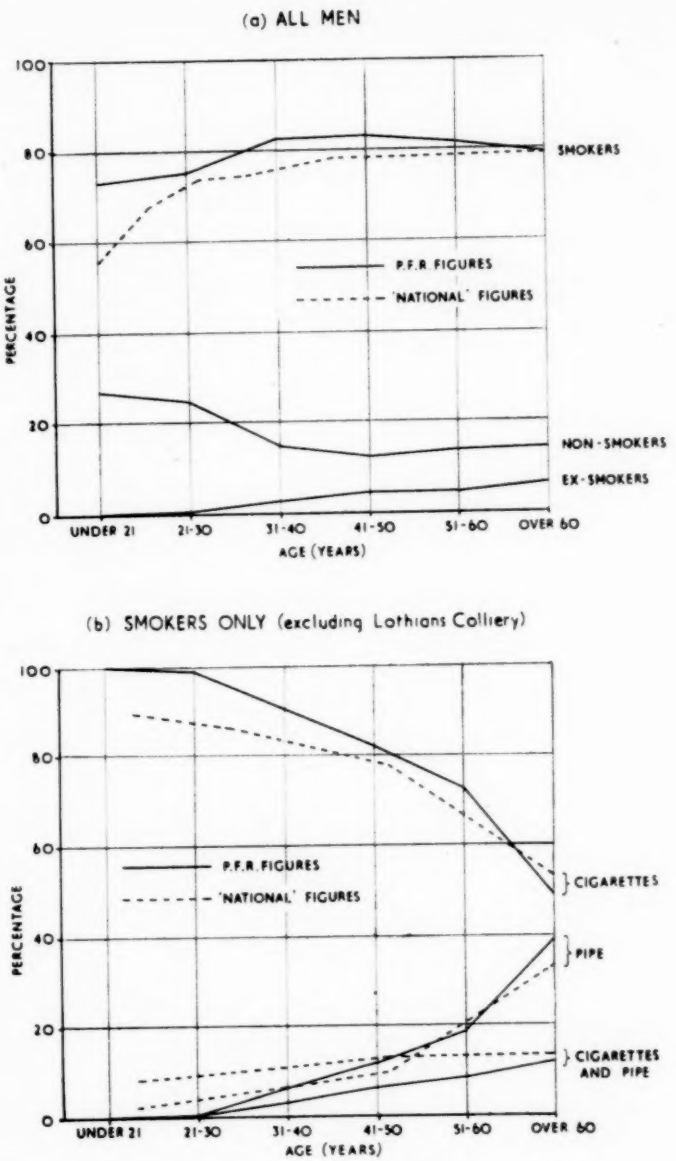

FIG. 1.—Smoking habits, by age.

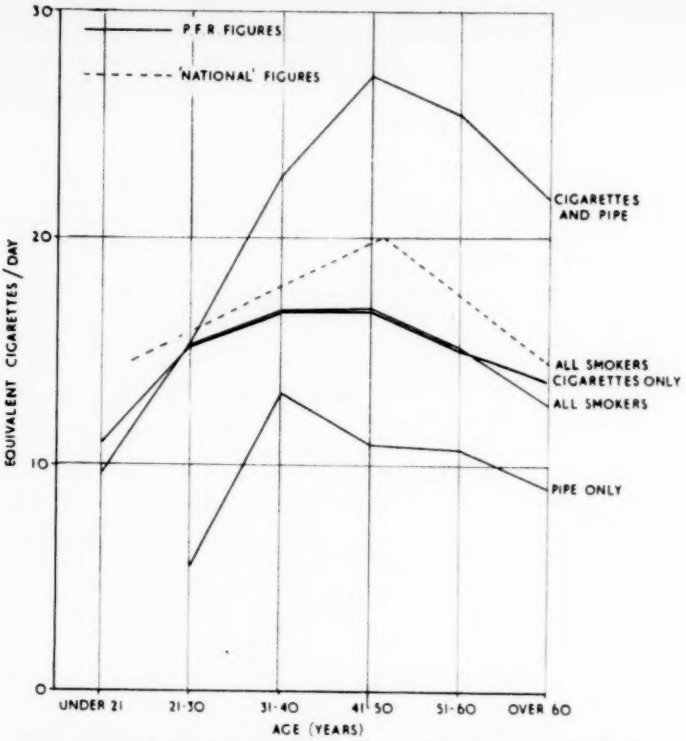

FIG. 2.—Amount of tobacco consumed, by age, at the Ayrshire and Lanarkshire collieries.

of the six age groups has been calculated for the smokers, ex-smokers, and non-smokers at the three collieries. The results for smokers at the Ayrshire and Lanarkshire collieries have been further subdivided in terms of the three smoking types and also into three classes according to the amount of tobacco smoked. Details are given in Table IV, which shows that the non-smokers and ex-smokers tend to be heavier than the smokers, the average difference being of the order of 3 to 4 kg. Age for age there is a tendency amongst the smokers for the weight of the men who consume the most tobacco to be above the

TABLE IV

AVERAGE WEIGHT (kg.), BY SMOKING HABITS AND AGE

Age Group (yrs)	Smokers							Non-smokers	Ex-smokers
	Type [All quantities]			Consumption (equivalent cigs/day) [All types]			All Smokers		
	Cigarettes* (only)	Pipe* (only)	Cigarettes* and Pipe	1–10*	11–20*	Over 20*			
Under 21	64·0	—	—	62·6	65·8	66·8	64·4	66·2	—
21–30	70·0	74·0	61·7	69·4	69·3	75·0	70·9	75·0	71·9
31–40	72·9	79·1	78·1	74·7	72·7	75·0	73·7	76·4	75·9
41–50	72·1	73·1	75·3	71·0	72·5	73·3	72·7	76·9	77·4
51–60	70·8	75·3	74·0	71·5	71·5	74·2	71·8	76·4	73·8
Over 60	69·8	77·0	76·9	74·4	72·7	72·3	72·3	77·0	76·8
All	70·7	75·5	75·3	70·3	71·4	74·0	71·7	74·6	75·9

* Excluding Lothians Colliery.

average although it is still not as high as for non-smokers or ex-smokers. However, when the type of smoking is considered, the cigarette smokers are, on average, about 5 kg. lighter than the pipe smokers and those smoking both cigarettes and pipe in all age groups. These latter smoking types are almost as heavy as the ex-smokers and very slightly heavier than the non-smokers.

The relation between smoking habits and sitting height is illustrated in Table V. In general, the average sitting height of the non-smokers and ex-smokers tends to be about 0·5 cm. greater than that of the smokers of the same age, and statistical tests show that this difference is significant. When the smokers are considered, there is a tendency for the sitting height of the men with the highest consumption of tobacco to be above the average for smokers generally, and, in fact, slightly greater than that of the non-smokers. The average sitting height of the pipe smokers does not differ consistently from that of the cigarette smokers of the same age, whereas that of the men smoking both cigarettes and pipe tends to be slightly higher.

Respiratory Symptoms

On the basis of the man's replies to the questionnaire an assessment was made of whether or not he had "respiratory symptoms" [*i.e.* bronchitis and/or asthma (Appendix)].

The results obtained at the three collieries are shown in Table VI (overleaf), and the general trends in Fig. 3, which indicates that the prevalence of the

TABLE V

AVERAGE SITTING HEIGHT (cm.), BY SMOKING HABITS AND AGE

Age Group (yrs)	Smokers							Non-smokers	Ex-smokers
	Type [All quantities]			Consumption (equivalent cigs/day) [All types]			All Smokers		
	Cigarettes* (only)	Pipe* (only)	Cigarettes* and Pipe	1–10*	11–20*	Over 20*			
Under 21	88·8	—	—	88·1	89·9	87·3	88·6	89·6	—
21–30	90·5	90·5	87·0	89·6	90·6	91·1	90·5	91·0	87·5
31–40	90·1	91·3	90·9	90·5	89·9	90·7	90·1	90·1	90·8
41–50	89·9	89·2	90·2	89·5	89·9	90·0	89·6	90·2	90·3
51–60	88·7	89·1	89·3	88·5	88·8	89·2	88·9	88·6	89·7
Over 60	87·9	88·2	89·7	87·9	88·3	89·6	87·7	88·5	88·3
All	89·6	89·2	89·8	88·9	89·7	90·1	89·5	90·0	89·9

* Excluding Lothians Colliery.

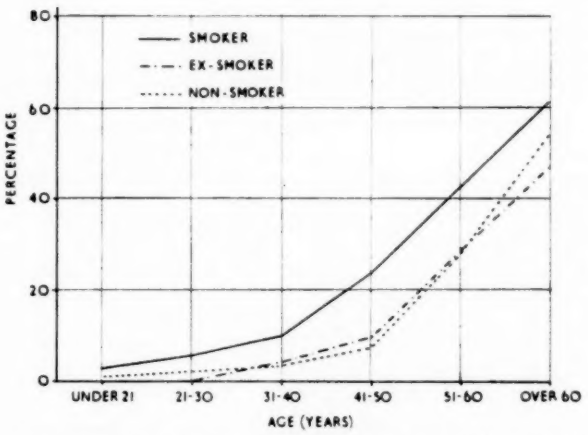

FIG. 3.—Prevalence of respiratory symptoms (per cent.), by age and smoking habits.

Age Group (yrs)	Type [All quantities]							
	Cigarettes* (only)			Pipe* (only)			Cigarette	
	Total No.	Men with R.S.		Total No.	Men with R.S.		Total No.	Men with R.S.
		No.	Per cent.		No.	Per cent.		No.
Under 21	149	5	3·4	0	0	—	0	0
21–30	295	12	4·1	2	0	0·0	1	0
31–40	338	27	8·0	23	2	8·7	12	1
41–50	368	99	26·9	52	8	15·4	28	5
51–60	292	135	46·2	75	28	37·3	34	18
Over 60	62	40	64·5	50	33	66·0	15	10
All	1,504	318	21·1	202	71	35·1	90	34

respiratory symptoms complex amongst the smokers is significantly higher than amongst the non-smokers and ex-smokers of the same age. When the smokers are considered separately, however, there is no evidence of any consistent relation between the prevalence of respiratory symptoms and amount of tobacco consumed. In the younger age groups, the lowest prevalence of respiratory symptoms is reported for the lightest smokers, but the reverse applies amongst the older men. Age for age there is no consistent difference in prevalence between the three types of smoker.

Ventilatory Function

The relation between F.E.V., age and smoking habits at the three collieries is illustrated in Table VII. In all groups there is a marked tendency for F.E.V. to decrease with age. The average F.E.V. of the non-smokers is slightly higher than that of the smokers, and the difference between the two groups is significant. The corresponding figures for the ex-smokers show no consistent pattern. Amongst the smokers, the average F.E.V. bears no consistent relationship to the amount of tobacco consumed or to the type of smoking.

When considering the difference in average F.E.V. between smokers and non-smokers, it must be borne in mind that there are significant differences in physique between the two groups. As it is found that sitting height is positively correlated with F.E.V., it is necessary to make some allowance for this factor as well as for age before any definite conclusions can be drawn. On the other hand, when account is taken of sitting height, there is no consistent relationship between weight and F.E.V. and the effect of weight can therefore safely be ignored. For each colliery separately, calculations were carried out to determine the relation between F.E.V., age, and sitting

Table VII

AVERAGE FEV.1 sec. (LITRES), BY SMOKING HABITS AND AGE

Age Group (yrs)	Smokers							Non-smokers	Ex-smokers
	Type [All quantites]			Consumption (equivalent cigs/day) [All types]			All Smokers		
	Cigarettes* (only)	Pipe* (only)	Cigarettes* and Pipe	1–10*	11–20*	Over 20*			
Under 21	3·87	—	—	3·74	4·02	4·17	3·81	4·00	—
21–30	3·87	3·80	2·30	3·83	3·90	3·72	3·84	3·96	3·58
31–40	3·39	3·57	3·27	3·50	3·38	3·36	3·40	3·46	3·72
41–50	2·88	2·76	2·97	2·89	2·89	2·80	2·92	3·03	2·96
51–60	2·35	2·48	2·29	2·28	2·41	2·37	2·42	2·55	2·59
Over 60	1·98	2·07	2·19	1·95	2·14	2·03	2·02	2·24	2·02
All	3·14	2·59	2·62	2·96	3·12	2·95	3·11	3·39	2·87

* Excluding Lothians Colliery.

SMOKING HABITS AND AGE

ers															
on (equivalent cigs/day) [All types]							All Smokers			Non-Smokers			Ex-Smokers		
10*		11–20*			Over 20*										
Men with R.S.		Total No.	Men with R.S.		Total No.	Men with R.S.		Total No.	Men with R.S.		Total No.	Men with R.S.		Total No.	Men with R.S.	
No.	Per cent.		No.	Per cent.		No.	Per cent.		No.	Per cent.		No.	Per cent.		No.	Per cent.
3	3·5	61	1	1·6	3	1	33·3	280	8	2·9	103	1	1·0	0	0	—
1	1·8	205	10	4·9	37	1	2·7	555	32	5·8	182	4	2·2	2	0	0·0
6	10·3	245	20	8·2	70	4	5·7	777	75	9·7	138	5	3·6	25	1	4·0
18	23·1	283	72	25·4	87	22	25·3	755	176	23·3	110	8	7·3	42	4	9·5
57	49·1	226	101	44·7	59	23	39·0	610	261	42·8	102	29	28·4	35	10	28·6
44	73·3	52	30	57·7	15	9	60·0	237	146	61·6	42	23	54·8	19	9	47·4
129	28·5	1,072	234	21·8	271	60	22·1	3,214	698	21·7	677	70	10·3	123	24	19·5

ding Lothians Colliery.

height. After due allowance had been made for age and sitting height, it was found that the F.E.V. of the smokers was still significantly lower than that of the non-smokers, the average difference being of the order of 0·1 litre. The results for all three collieries together are summarized in Table VIII. There was no evidence of any systematic relationship between age and the difference in F.E.V. between smokers and non-smokers for men less than 50 years old. Amongst the oldest members of the population the difference between smokers and non-smokers was appreciably greater than amongst the remainder of the population. This may, however, be associated with retirement amongst the older men, who thereby form a more highly selected population than the younger age groups.

TABLE VIII

AVERAGE F.E.V.$_{1\,sec.}$ (LITRES) OF SMOKERS AND NON-SMOKERS BY AGE SHOWING EFFECT OF CORRECTION FOR SITTING HEIGHT

Age Group (yrs)	Before Correction for Sitting Height			After Correction for Sitting Height		
	Non-smokers	Smokers	Difference	Non-smokers	Smokers	Difference
Under 21	4·00	3·81	0·19	4·09	3·96	0·13
21–30	3·96	3·84	0·12	3·86	3·77	0·09
31–40	3·46	3·40	0·06	3·44	3·38	0·06
41–50	3·03	2·92	0·11	3·04	2·96	0·08
51–60	2·55	2·42	0·13	2·71	2·56	0·15
Over 60	2·24	2·02	0·22	2·38	2·21	0·17

RADIOLOGICAL PNEUMOCONIOSIS

As the numbers of men assigned to the higher categories of pneumoconiosis at the three collieries are small, the prevalence of pneumoconiosis has been expressed in terms of only two classes—Category 0 and Category 1 or more. Details of the relationship between radiological pneumoconiosis and smoking habits are given in Table IX (overleaf). In general, there is no consistent difference between the prevalence of pneumoconiosis in smokers and non-smokers in the various age groups, but for both smokers and non-smokers it tends to be rather lower than for ex-smokers. When the smokers are considered separately, the amount of tobacco consumed by the younger men bears no consistent relationship to the prevalence of penumoconiosis. For men more than 50 years old, however, the prevalence is considerably lower amongst those who smoke most than amongst those who smoke least. There is no consistent difference between the prevalence of pneumoconiosis in the various age groups amongst the three smoking types.

DISCUSSION

The information about smoking habits quoted in this paper is based on four short questions forming part of the respiratory symptoms questionnaire which is asked of each man during his medical examination. The results obtained were fairly consistent with those reported in other inquiries, and it is considered that they probably provide a valid measure of the true situation. In comparison with the figures given by Todd (1957) for all males in the United Kingdom, the mining populations under consideration included a higher proportion of men smoking cigarettes only and a lower proportion of

PREVALENCE OF CATEGORY 1 OR MORE PNEUMO

Age Group (yrs)	Type [All quantities]								
	Cigarettes* (only)			Pipe* (only)			Cigarette		
	Total No.	Men with Pn.		Total No.	Men with Pn.		Total No.	Men with Pn.	
		No.	Per cent.		No.	Per cent.		No.	
Under 21	149	0	0·0	0	0	—	0	0	
21–30	295	0	0·0	2	0	0·0	1	0	
31–40	338	7	2·1	23	0	0·0	12	0	
41–50	368	38	10·3	52	4	7·7	28	1	
51–60	292	127	43·5	75	33	44·0	34	16	
Over 60	62	42	67·7	50	32	64·0	15	9	
All	1,504	214	14·2	202	69	34·2	90	26	

men smoking both cigarettes and pipe. The total consumption of tobacco by the smokers was slightly below the national average. This is not surprising, as the underground workers amongst the men under observation were not allowed to smoke at their place of employment.

At each colliery the prevalence of respiratory symptoms, as judged by the specified combination of positive answers to questions about cough, phlegm, etc., was found to be appreciably higher amongst the smokers than that amongst the non-smokers in all age groups. The tendency amongst the younger men for the difference between smokers and non-smokers to increase with age (Table VI) suggests that there is a direct association between smoking and respiratory symptoms. The differences in mean weights between the two groups also increased with age (Table IV), the smokers being lighter.

On the other hand, although there was a significant difference between smokers and non-smokers as regards sitting height (Table V), the difference did not increase systematically with age. This suggests that the difference here is more likely to be due to a population selection effect than to a direct association with smoking. Thus, although the men who smoked most of all were comparable in height with the non-smokers, the average smoker was shorter. Similarly, the lowering of forced expiratory volume recorded for smokers, even after correction for height, remains reasonably uniform with age up to about 50 years (Table VIII). This also suggests a population selection effect rather than an effect due to smoking, which would be expected to increase with prolongation of the habit. The possibility that smokers are a self-selected group is consistent with the findings of Eysenck, Tarrant, Woolf, and England (1960), who demonstrated differences in certain personality traits between smokers and non-smokers which they attributed to genotypic differences. There is, however, a possible alternative explanation in that smoking in youth might retard physical development, especially ventilatory function (the majority of the men classed as smokers probably started smoking in their youth), whereas once growth has been completed smoking has no further effect.

The average difference in F.E.V. between smokers and non-smokers in the population examined was 0·1 litre, or about 3 per cent., after allowance had been made for sitting height and age. Comparison of this result with those of other workers is complicated by several factors, such as variations in the method of measuring ventilatory capacity, in the definition of "non-smokers", and in age distribution and smoking habits amongst the various populations examined. For example, most other investigators have dealt with smaller numbers, especially of non-smokers, and have reported the mean indirect maximum breathing capacity as their measure of ventilatory capacity, frequently using the $F.E.V._{0\cdot75\,sec.}$ as the basis of calculation. They have also usually refrained from making allowance for height. However, bearing these differences in mind, it is possible to compare some results in terms of the percentage lowering of ventilatory capacity, rather than of the absolute lowering, associated with smoking. Thus Olsen and Gilson (1960), in a population of male non-miners aged 55 to 64 years in Bornholm, found a difference of 14 per cent. (106·2 to 91·1 l./min.) in the mean

SMOKING HABITS AND AGE

ers	Consumption (equivalent cigs/day) [All types]								All Smokers			Non-Smokers			Ex-Smokers		
	1–10*			11–20*			Over 20*										
Cigarettes	Men with Pn.		Total No.	Men with Pn.		Total No.	Men with Pn.		Total No.	Men with Pn.		Total No.	Men with Pn.		Total No.	Men with Pn.	
	No.	Per cent.		No.	Per cent.		No.	Per cent.		No.	Per cent.		No.	Per cent.		No.	Per cent.
0	0	0·0	61	0	0·0	3	0	0·0	280	0	0·0	103	0	0·0	0	0	—
0	0	0·0	205	0	0·0	37	0	0·0	555	0	0·0	182	1	0·5	2	0	0·0
0	0	0·0	245	6	2·4	70	1	1·4	777	14	1·8	138	0	0·0	25	1	4·0
1	6	7·7	283	26	9·2	87	11	12·6	755	62	8·2	110	10	9·1	42	4	9·5
16	53	45·7	226	99	43·8	59	24	40·7	610	235	38·5	102	32	31·4	35	16	45·7
9	40	66·7	52	35	67·3	15	8	53·3	237	131	55·3	42	29	69·0	19	9	47·4
26	99	21·9	1,072	166	15·5	271	44	16·2	3,214	442	13·8	677	72	10·6	123	30	24·4

ding Lothians Colliery.

indirect M.B.C. between 29 non-smokers and 13 men who smoked cigarettes only (average consumption 12 g./day). Fletcher and others (1959), in their population of London postmen aged 40 to 59 years, found differences between eight non-smokers (mean indirect M.B.C., standardized to age 40, 132·8 l./min.), 87 smokers with a tobacco consumption of 1 to 14 g./day (110·9 l./min.), and 37 smokers with a consumption of 15 g. or more/day (104·3 l./min.), corresponding to a 17 per cent. drop in ventilatory capacity for the "light" smokers and of 21 per cent. for the "heavy" smokers. Higgins (1959), studying a random selection of 734 men aged 25–64 years, including non-miners as well as miners and ex-miners in urban and rural populations, reported a weighted difference of 9 l./min. (about 9 per cent.) between non-smoker and smokers, which was not due to small differences in the age distributions between the two groups. All these results show higher percentage differences between smokers and non-smokers than those summarized in Table VIII (even before correcting for height). It might therefore appear that smoking is associated with a smaller reduction in ventilatory capacity amongst coalminers than amongst non-miners. This is not impossible, when it is remembered that the majority of coalminers are employed underground, where they are not permitted to smoke. This means that their smoking is done during their leisure hours, and not when they are hard at work. It is, of course, impossible to investigate this factor using the data presented here, and relatively little information has been published in the literature. Higgins (1959) has examined comparatively small groups of men and finds, for example, a difference between non-smokers and smokers of about 10 per cent. (100 to 90 l./min.) for non-miners aged 55 to 64, compared with about 10 per cent. for miners (including ex-miners) without radiological pneumoconiosis and 20 per cent. for miners with simple pneumoconiosis. In the 25 to 34-year age group, his reported differences are about 8 per cent. for non-miners (in the Rhondda Fach), 15 per cent. for miners (including ex-miners) with Category 0 pneumoconiosis, and −10 per cent. for miners with Category 3 pneumoconiosis. For a corresponding group of non-miners aged 35 to 54 years, the value is about 20 per cent. The variations in these figures, both amongst themselves and in comparison with those quoted in Table VIII, are probably due to the small numbers of non-smokers in the various groups. This precludes the drawing of definite conclusions about the possible difference in the effect of smoking on ventilatory function in coalminers and non-miners. However, the subject is of considerable interest and worthy of further study.

It is interesting to note that, although differences in physique and ventilatory function have been found between smokers and non-smokers, the results obtained for the smokers show relatively little variation with the amount of tobacco currently consumed. Todd and Laws (1958) have observed that there may be considerable variations in individual smoking habits even within a short space of time, and they suggest that figures for the current consumption of tobacco by smokers cannot reasonably be extended to the whole of their past history. This conclusion is supported by the absence in these results, as in those of Higgins (1959), of any

consistent relation between physique or ventilatory function and current consumption of tobacco. The only appreciable difference between the men smoking cigarettes only, pipe only, and cigarettes and pipe was noted in connexion with weight, which was considerably lower amongst the cigarette smokers.

The prevalence of pneumoconiosis tends to be higher amongst ex-smokers than amongst smokers and non-smokers, except in the oldest age group. Amongst smokers of 50 years and above, the prevalence of pneumoconiosis is lowest amongst the men who smoke the most and highest amongst the men who smoke the least. Both these effects might be due to a tendency for the men with pneumoconiosis to reduce their tobacco consumption.

SUMMARY

The relation between smoking habits and physique, respiratory symptoms, ventilatory function, and radiological pneumoconiosis amongst the underground and surface workers at three Scottish collieries has been investigated, using data obtained during the medical surveys carried out by the National Coal Board's Pneumoconiosis Field Research. In general, the information obtained about current smoking habits follows a similar pattern to that reported on other recent surveys, although the observed proportion of smokers was slightly greater and the total consumption of tobacco by smokers was slightly less than the average recorded in a recent survey of males on a national basis. When the relation between physique and smoking habits was considered, it was found that in all age groups the non-smokers were, on average, taller and heavier than the smokers. The prevalence of respiratory symptoms amongst the non-smokers was appreciably lower than that amongst smokers of the same age. The ventilatory function (as measured by the F.E.V.1 sec.) of the non-smokers in all age groups was found to be significantly higher than that of the smokers, even when account was taken of differences in physique and age. In contrast to the differences between smokers and non-smokers, no consistent relationship was recorded between current consumption of tobacco and physique, respiratory symptoms, or ventilatory function amongst the smokers.

Some evidence was found which was consistent with a tendency for men with pneumoconiosis to reduce their tobacco consumption.

This paper is published by permission of the National Coal Board, who are not necessarily committed to the views expressed. The work forms part of the Board's Pneumoconiosis Field Research. Our thanks are due to Dr. J. M. Rogan (Chief Medical Officer, National Coal Board), under whose direction this work was carried out, to Drs. S. Rae and P. J. Chapman, who read the films, and to Mr. D. Hicks for invaluable discussions and advice.

REFERENCES

Abbott, O. A., Hopkins, W. A., Van Fleit, W. E., and Robinson, J. S. (1953). *Thorax*, **8**, 116.

Ashford, J. R., Forwell, G. D., and Routledge, R. (1960). *Brit. J. industr. Med.*, **17**, 114.

Bickerman, H. A., and Barach, A. L. (1954). *J. Lab. clin. Med.*, **43**, 455.

Brown, R. G., McKeown, T., and Whitfield, A. G. W. (1957). *Brit. J. prev. soc. Med.*, **11**, 162.

Carpenter, R. G., Cochrane, A. L., Gilson, J. C., and Higgins, I. T. T. (1956). *Brit. J. industr. Med.*, **13**, 166.

Clifton, M. (1956). *Trans. Ass. industr. med. Offrs*, **6**, 66.

Doll, R., and Hill, A. B. (1950). *Brit. med. J.*, **2**, 739

Eich, R. H., Gilbert, R., and Auchincloss, J. H. (1957). *Amer. Rev. Tuberc.*, **76**, 22.

Eysenck, H. J., Tarrant, M., Woolf, M., and England, L. (1960). *Brit. med. J.*, **1**, 1456.

Fay, J. W. J. (1957). *Nature (Lond.)*, **180**, 309.

—— and Rae, S. (1959). *Ann. occup. Hyg.*, **1**, 149.

Fletcher, C. M., Elmes, P. C., Fairbairn, A. S., and Wood, C. H. (1959). *Brit. med. J.*, **2**, 257.

Franklin, W. (1958). *J. clin. Invest.*, **37**, 895.

Gaensler, E. A. (1951). *Amer. Rev. Tuberc.*, **64**, 256.

Higgins, I. T. T. (1959). *Brit. med. J.*, **1**, 325.

——, Cochrane, A. L., Gilson, J. C. and Wood, C. H. (1959). *Brit. J. industr. Med.*, **16**, 255.

——, Oldham, P. D., Cochrane, A. L. and Gilson, J. C. (1956). *Brit. med. J.*, **2**, 904.

International Labour Office, Geneva (1953). "Proceedings of the 3rd International Conference of Experts on Pneumoconiosis". Sydney, 1950.

Ogilvie, A. G., and Newell, D. J. (1957). "Chronic Bronchitis in Newcastle-upon-Tyne". Livingstone, Edinburgh and London.

Olsen, H. C., and Gilson, J. C. (1960). *Brit. med. J.*, **1**, 450.

Palmer, K. N. V. (1954). *Ibid.*, **1**, 1473.

Rae, S. (1960). Private communication.

Rogan, J., Ashford, J. R., Chapman, P. J., Duffield, D. P., Fay, J. W. J., and Rae, S. (1961). *Brit. med. J.*, 1, 1337.

Todd, G. F. (1957). Tobacco Manufacturers' Standing Committee Research Papers. No. 1, "Statistics of Smoking".

—— and Laws, J. T. (1958). Tobacco Manufacturers' Standing Committee Research Papers, No. 2, "The Reliability of Statements about Smoking Habits".

APPENDIX

Respiratory Symptoms Questionnaire

PREAMBLE

I am going to ask you some questions about your chest, about cough and spit, for example. Please try and answer "yes" or "no", but if you do not understand a question then ask me about it.

Cough		
Do you cough when you get up or first thing in the morning?	Yes/No	1
Do you cough during the rest of the day—I don't mean at the end of the shift?	Yes/No	2
If the answer to 2 is "yes" go on to 3 If "no" go on to 4		
Do you cough like this on most days for as much as 3 months in the year?	Yes/No	3
Phlegm		
Do you bring up phlegm when you get up or first thing in the morning?	Yes/No	4
Do you bring up phlegm during the rest of the day?	Yes/No	5
If the answer to 5 is "yes" go on to 6 If "no" go on to 7		
Do you bring up phlegm like this on most days for as much as 3 months in the year? ...	Yes/No	6
Breathlessness		
Do you have to walk slower than other people on the level?	Yes/No	7
Wheezing		
Do you ever have wheezing or whistling in your chest?—I don't mean when you have a cold	Yes/No	8
Weather		
Does the weather affect your chest?	Yes/No	9
Smoking		
Do you smoke?	Yes/No	10
If yes: How many cigarettes per week	.....	11
How many oz. tobacco per week	.....	12
If no: Have you ever smoked as much as ten cigarettes a day for 10 years?... ...	Yes/No	13
Chest Illness		
In the last 3 years have you had a chest illness that has kept you off work for more than a week?	Yes/No	14
If yes: What did your doctor say it was?	.....	15

RESPIRATORY SYMPTOMS TO BE DIAGNOSED IF POSITIVE ANSWERS ARE GIVEN TO:

(*a*) QUESTIONS 3 OR 6 **AND** 7 OR 8 OR 9 OR 14

OR

(*b*) QUESTIONS 8 AND 9

Brit. J. prev. soc. Med. (1961), **15**, 118

MULTIPLE SCLEROSIS IN BRITISH COMMONWEALTH COUNTRIES IN THE SOUTHERN HEMISPHERE

BY

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In the United States of America, which extend from approximately 26° to 49° North, the suggestion that a four-fold increase in the frequency of multiple sclerosis occurred between 30° and 40° was originally based on crude mortality data (Limburg, 1950). Recent detailed field studies have confirmed this impression (Kurland, 1952; Alter, Allison, Talbert, and Kurland, 1960; Kurland, Mulder, and Westlund, 1955). In ex-servicemen of the U.S. Armed Forces, a close correlation has also been demonstrated between the latitude of their place of birth and the frequency with which they subsequently developed miltiple sclerosis (Acheson, Bachrach, and Wright, 1960). This relationship appears to hold not only for white veterans but for Negroes (Acheson and Bachrach, 1960). All North American studies agree that the disease is rare in the states of the deep South (Limburg, 1960; Kurland, 1952; Alter and others, 1960). In Europe, multiple sclerosis is certainly at least as prevalent in the British Isles (Allison and Millar, 1954; Miller, Ridley, and Schapira, 1960; Sutherland, 1956) and the northern and central Continental countries (Hyllested, 1956; Bammer and Schaltenbrand, 1960; Dassel, 1960; Georgi and Hall, 1960) as in the Northern States of America, and may be less frequent along the Mediterranean littoral. These differences have been interpreted as suggesting that there is an important, but as yet unknown, environmental factor correlated with latitude in the aetiology of multiple sclerosis (Kurland and others, 1955; Acheson and Bachrach, 1960). The alternative view that the geographical distribution is due to differences in racial susceptibility, or to differences in diagnostic standards, is becoming more difficult to maintain, particularly in regard to the distribution of the disease in the United States.

If an environmental factor which varies with latitude is important in multiple sclerosis, it should be possible to demonstrate a fluctuation in the frequency of the disease within the settler populations of the southern hemisphere. Indeed the white people of New Zealand, Australia, and South Africa are particularly suitable for such a study, because an overwhelming majority of their forebears came from susceptible stock from the British Isles or Holland (50°–60°N.) whereas their descendants now live over a much wider range of latitude in the southern hemisphere (from 22°–47°S.). This being so, if the environmental hypothesis is correct, we should expect the disease to diminish from a frequency somewhat less than that of England and Wales (mean latitude 52·5°N.) in the South Island of New Zealand (mean latitude 44°S.) to extreme rarity in the Transvaal and Orange Free State. In the present paper mortality data has in general been used, as apart from Dean's preliminary report from South Africa (Dean, 1949) information about morbidity is not yet available. For New Zealand, however, hospital discharge rates have also been obtained. Some comparisons have been made between the multiple sclerosis data and the death rates experienced in these countries from amyotrophic lateral sclerosis.

Material

Information was obtained from the Departments of Medical Statistics of the various countries concerning deaths attributed to rubrics 345 (multiple sclerosis) and 356 (motorneurone disease; amyotrophic lateral sclerosis) of the Sixth Revision of the International Statistical Classification (W.H.O., 1948) during the years 1950–9 for the States of Australia, the Islands of New Zealand, and the Union of South Africa. Only data concerning whites were considered. As the populations concerned are small, average death rates for the decade were computed from the 1950–9 aggregates, using the best estimates

available for the white populations for 1954. Similar data for the years 1954–6 were collected for the countries of origin of the white stock under study, namely England and Wales, Scotland, and Northern Ireland. The proportion of all deaths over the age of 65 attributed to "senility, senile decay, etc., without mention of psychosis" (rubric 794 I.S.C.) was used as a rough index of the standard of death certification in the countries under study.

RESULTS

Fluctuation with Latitude and with Sunshine. — Estimates were made of the mean latitudes of the populations under study in the following manner. In Australia, where a little more than half of the population lives within the metropolitan areas of the state capitals, the latitude of these capitals were taken as representative of the bulk of the population of the six states. For New Zealand, where the bulk of the population is clustered at the extremities of the two islands, the mean latitude of the North Island was estimated as 39°S. and of the South Island 44°S. For South Africa, where the principal white population centres of the Cape (33·5°) and the Rand (26°) are widely separated, 29·5°S. was taken as a representative figure for the whole white population. For England and Wales, Scotland, Northern Ireland, and the Irish Republic, the latitudes of Birmingham, Edinburgh, Belfast, and Dublin respectively were used. For the four Census Regions of the United States and for Canada, the representative places taken were Atlanta (South), San Francisco (West), Chicago (North Central), New York (North East), and Ottawa. These estimates are probably correct within half a degree. For Australia, New Zealand, South Africa, and the United Kingdom, estimates of the average total annual hours of sunshine at the points stated (as measured by the Campbell-Stokes instrument) were also obtained (Brooks, 1950; Garnier, 1958; Government of S. Africa; Annual Abstract of Statistics, 1958). North American figures could not be used as they are not recorded in a comparable way (Gorczynski, 1948).

The average age-adjusted death rates from multiple sclerosis and amyotrophic lateral sclerosis are shown in the Table. It can be seen at once that, whether taken individually or as a group, the death rates from multiple sclerosis in the countries of origin (British Isles) are higher than in any of the white settler populations of the southern hemisphere. For motorneurone disease, on the other hand, there is no gross difference between the British age-adjusted

TABLE

AVERAGE AGE-ADJUSTED DEATH RATES FROM MULTIPLE SCLEROSIS AND AMYOTROPHIC LATERAL SCLEROSIS

Country	Estimated Population (thousands)	Average Total Annual Hours of Sunshine	Mean Latitude (°)	Multiple Sclerosis			Amyotrophic Lateral Sclerosis			Proportion of Deaths over 65 attributed to Senility within the Same Years
				Deaths	Years	Average Age-adjusted Death Rate	Deaths	Years	Average Age-adjusted Death Rate	
England and Wales	44,623	1,445	52·5°N	2,453	1954–6	1·60	1,537	1954–6	0·92	2·4
Scotland	5,133	1,220	56°N	488	1954–6	2·93	199	1954–6	1·14	2·1
N. Ireland	1,393	1,285	54·5°N	128	1954–6	3·14	32	1954–6	0·70	—
UNITED KINGDOM	51,149	—	—	3,069		1·77	1,768		0·94	—
New South Wales	3,424	2,475	34°S	223	1950–9	0·65	326	1950–9	0·95	3·3
Victoria	2,452	2,225	37·5°S	237	1950–9	0·96	254	1950–9	1·01	1·8
Queensland	1,318	2,700	27·5°S	94	1950–9	0·76	115	1950–9	0·90	2·7
S. Australia	797	2,550	35°S	52	1950–9	0·67	84*	1953–9*	1·45	2·8
W. Australia	639	2,810	32°S	22	1950–9	0·37	50	1950–9	0·82	0·7
Tasmania	309	2,120	42°S	35	1950–9	1·25	25	1950–9	0·89	1·5
AUSTRALIA	8,986	—	—	663		0·74	890		1·00	2·5
North Island	1,283	2,050	39°S	130	1950–9	1·02	170	1950–9	1·25	1·2
South Island	646	1,850	44°S	81	1950–9	1·31	84	1950–9	1·20	0·9
NEW ZEALAND	1,929	—	—	211		1·16	254		1·24	1·1
UNION OF SOUTH AFRICA	2,642	3,000	29·5°S	22	1950–8	0·09†	68	1950–8	0·31	—

*1950–2 figures not available. An estimate of 120 deaths ($84 \times \frac{10}{7}$) was therefore used in computing the average annual death rate for the whole of Australia, 1950–59.

†Provincial death rates: Natal 0·2; Cape 0·07; Transvaal and Orange Free State 0·06.

Notes: The figures for Australia, New Zealand, and South Africa are for the white population. For Australia no deaths from multiple sclerosis or amyotrophic lateral sclerosis were reported within the small populations of the Northern Territory or the Australian Capital Territory.

rate* (0·94 per 100,000) and the New Zealand and Australian rates (1·24 and 1·00 per 100,000). The South African figure is an exception and is lower.

The age-adjusted death rates for multiple sclerosis and motorneurone disease was plotted against latitude in Fig. 1. For the northern hemisphere (circles), in addition to the United Kingdom, figures for the Irish Republic (W.H.O., 1956) (dotted circles), the regions of the United States (whites), and Canada (Kurland, 1952) are also given. The figures for the Irish Republic have not been used in the regression as information about the current coding policy for deaths attributed to cerebral sclerosis, etc., was not obtained. In the northern hemisphere the correlation between the multiple sclerosis rates and latitude is highly significant ($r = +0·90$; $·001 < p < ·01$) and a similar degree of correlation is present for the states (dots) in the southern hemisphere ($r = +0·86$; $·001 < p < ·01$). The line drawn in Fig. 1(*a*) represents the regression equation for the northern hemisphere, and it is striking how well it fits the values for the southern hemisphere. The steep gradient of the line is also notable, indicating an increase in reported death rate of 1·05 per 100,000 per 10° of latitude. Queensland is the only striking exception to the general trend.

For motorneurone disease the situation is different. In the northern hemisphere (circles) there is a relationship with latitude ($r = +0·79$; $p = 0·02$). However, the gradient is slight and there is an increase in death rate of only 0·16 per 100,000 per 10° of latitude. Similar correlations and gradients can be shown for other diseases within the regions of the United States, Canada, and England and Wales, notably regional enteritis (Acheson, 1959, p. 481), ulcerative colitis (Acheson, 1959, p. 469) and Parkinsonism (Kurland, 1959), and these are probably related to differences in the standard of death certification from South to North within these countries. For the

*All death rates were adjusted to the structure of the United States population in 1950.

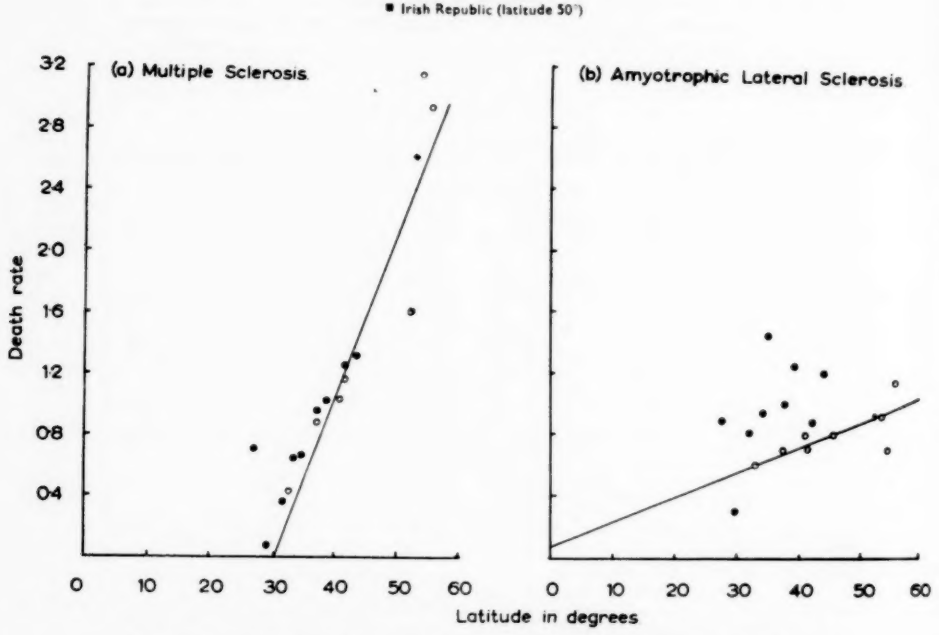

FIG. 1.—Average death rates by mean latitude. (*a*) multiple sclerosis (*b*) amyotrophic lateral sclerosis.

(*a*) The equation of the line (average age-adjusted death rate for multiple sclerosis, regions of the U.S., Canada, England and Wales, Scotland, and Northern Ireland is $y = 0·105x - 3·196$.
(*b*) The equation for the corresponding line for amyotrophic lateral sclerosis is $y = 0·0159x + 0·0755$.

southern hemisphere (dots), however, there is no significant correlation between mortality from motorneurone disease and latitude ($r = +0{\cdot}51$; $p > 0{\cdot}1$), although a trend is present in this direction. It is also noteworthy that the line drawn in Fig. 1(*b*) for the northern hemisphere does not describe the data from the southern hemisphere.

The proportion of deaths over the age of 65 attributed to the unsatisfactory diagnosis "senility or senile decay without mention of psychosis" (Table I) was taken as a rough index of the standard of death certification in each country. It was found that there was a tendency for the States of Australia nearer the Equator to have a higher proportion of deaths certified under this heading than the more southerly states. This suggests an inverse relationship between standard of death certification and latitude such as can be demonstrated by using the same criteria within the United States (U.S. Dept. of Health). In fact, this relationship is not at all well-marked within the countries under study in the southern hemisphere. Thus, in New Zealand, the proportions for the two islands are not significantly different; and, for the components of Australia and New Zealand together, the correlation coefficient (proportion of deaths over 65 years ascribed to senility versus latitude) is only $-0{\cdot}51$ ($p > 0{\cdot}1$).

When the death rates ascribed to multiple sclerosis, amyotrophic lateral sclerosis, and "senility" were correlated with the average total annual hours of sunshine, a highly significant negative correlation (the more sunshine received, the lower the death rate experienced) was obtained for multiple sclerosis ($r = -0{\cdot}95$; $p < 0{\cdot}001$) but not for amyotrophic lateral sclerosis ($r = -0{\cdot}62$; $0{\cdot}05 < p < 0{\cdot}1$). For "senility" there was no material trend ($r = +0{\cdot}39$).

Age- and Sex-Specific Rates. — The average age-specific death rates from multiple sclerosis by sex for Australia, New Zealand, and England and Wales are shown in Fig. 2. (The number of deaths reported from South Africa is too small to be handled in this way.) The graphs for England and Wales and New

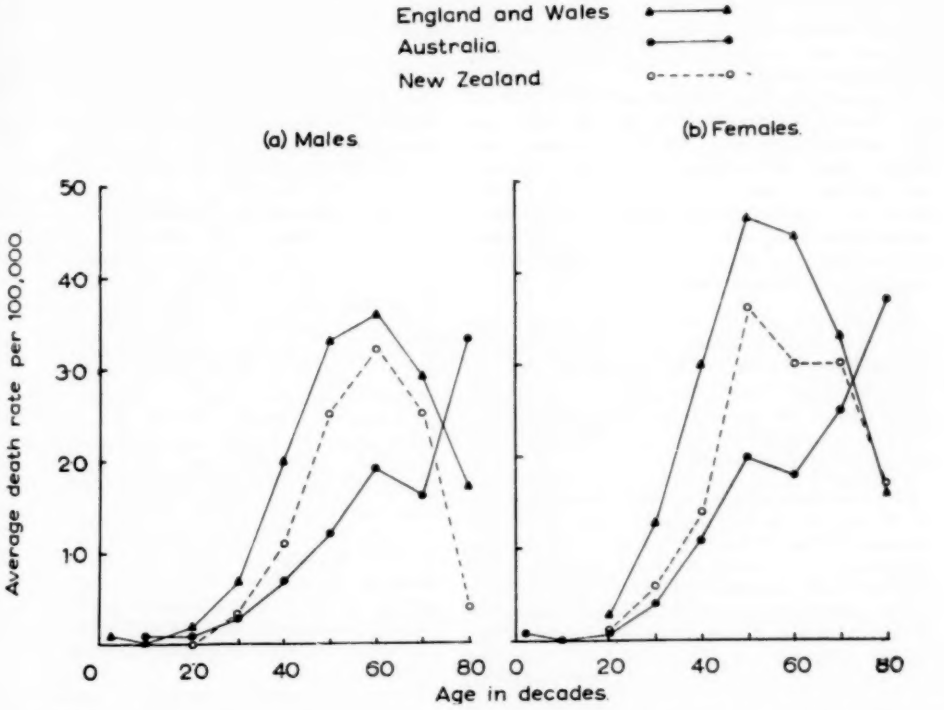

FIG. 2.—**Average age-specific death rates from multiple sclerosis, by sex for England and Wales (1954–5), New Zealand (1950–9), and Australia (1950–9).**

Zealand are strikingly similar in form, with peaks in the 45 to 54 year age group in women and the 55 to 64 year age group in men. The symmetrical form of these curves closely resembles the age-specific prevalence curves found in recent American (Kurland, 1952; Alter and others, 1960) and British (Allison and Millar, 1954) surveys, although the peaks of prevalence fall, as expected, 10 to 20 years earlier. The age-specific curves for Scotland and Northern Ireland, which are not shown here because of lack of space, also exhibit a peak in middle age followed by a decline. The Australian age-specific curves are biphasic, with the first peak at 45 to 54 years and 55 to 64 years for females and males respectively, followed by a slight fall, and then by a second steep rise in old age. Such curves suggest an admixture of two disease entities. It is possible that, in spite of the recent change in death certification procedure*, there is still some confusion in the classification of cerebral arteriosclerosis and multiple sclerosis. A much less likely alternative is that the age-pattern of deaths due to multiple sclerosis differs in Australia. The published curves for the United States and Canada show a steady increase with age and resemble the Australian curve more closely than those for New Zealand and England and Wales.

A consistent female:male ratio of 1·4:1 was found when the average sex-specific death rates from multiple sclerosis were compared for Australia, New Zealand, South Africa, England and Wales, Scotland, and Northern Ireland. Such an effect was not observed for mortality from multiple sclerosis in the United States or Canada, but recent American morbidity studies have agreed that the prevalence of the conditions is higher in women than in men.

Secular Trend.—Within the period 1950–59, there has been no consistent trend in the crude death rate from multiple sclerosis in New Zealand or Australia. For South Africa the reported deaths are too few to permit annual rates to be compared but there appears to have been an increase in the mortality ascribed to multiple sclerosis since 1956.

Morbidity Data.—In New Zealand the vast majority of all hospital admissions take place within the National Service, which operates to an equal extent throughout both Islands. A central diagnostic index is kept. When duplicate admissions for the same person had been eliminated, it was found that, within the years 1950–7, a total of 617 white persons had been discharged from or had died in New Zealand public hospitals with multiple sclerosis, an overall hospitalization rate of 4 per 100,000 per annum. In the same period nine Maoris had been hospitalized, giving an average annual rate of 0·9 per 100,000. When the European rates for the North and South Islands were considered separately, it was found that the average annual discharge rate for the South Island ($5·0 \pm 0·31$ per 100,000) exceeded that for the North Island ($3·5 \pm 0·19$ per 100,000). This difference is statistically significant ($·001 < p < ·01$). The age-specific discharge rates by sex for each island are seen in Fig. 3 (opposite).

Discussion

The object of this paper has been to test in the southern hemisphere the hypothesis derived from data from the northern hemisphere that, within peoples of European stock, the frequency of multiple sclerosis is related to latitude. If mortality data represent an index of the frequency of multiple sclerosis, the hypothesis has withstood the test. Thus, not only has a correlation with latitude of similar magnitude been demonstrated for the southern hemisphere, but the regression equation for the northern hemisphere (which states the hypothesis under examination in mathematical form) adequately describes the data for the southern hemisphere with the exception of Queensland.

Unfortunately, in chronic diseases, it cannot be assumed that mortality is an index of morbidity without careful scrutiny. Hyllested (1956) has pointed out that in Denmark only about half of the known cases of multiple sclerosis are at death certified as having died of that disease. On the other hand, Sutherland (1956) in Scotland, found slightly more deaths certified than he would have expected from his field study. Kurland and Moriyama (1951), in a survey of death certificates from the United States and Canada, found that in 1946 the death rates attributed to multiple sclerosis were inflated by about 20 per cent. with cases of "cerebral arteriosclerosis". Fortunately this term and the other ambiguous term "general sclerosis" is reported as being used rarely (less than 2 per cent. of all deaths in rubric 345) in the British Isles, and since 1950 this has been the subject of further inquiry by the Registrars of all countries under study except South Africa. However, it is prudent to assume that the mortality ascribed to multiple sclerosis is still subject to large errors of both under-reporting and misreporting.

In spite of these acknowledged errors, Limburg (1950) drew the conclusion from crude mortality data that multiple sclerosis became more frequent

*In Australia, New Zealand, and England and Wales (Registrars General) "cerebral sclerosis" and "general sclerosis" are now coded to arterio-sclerosis unless the certifying practitioner indicates that multiple sclerosis was meant. In Northern Ireland and Scotland the Registrars report that these terms are very rarely used on death certificates.

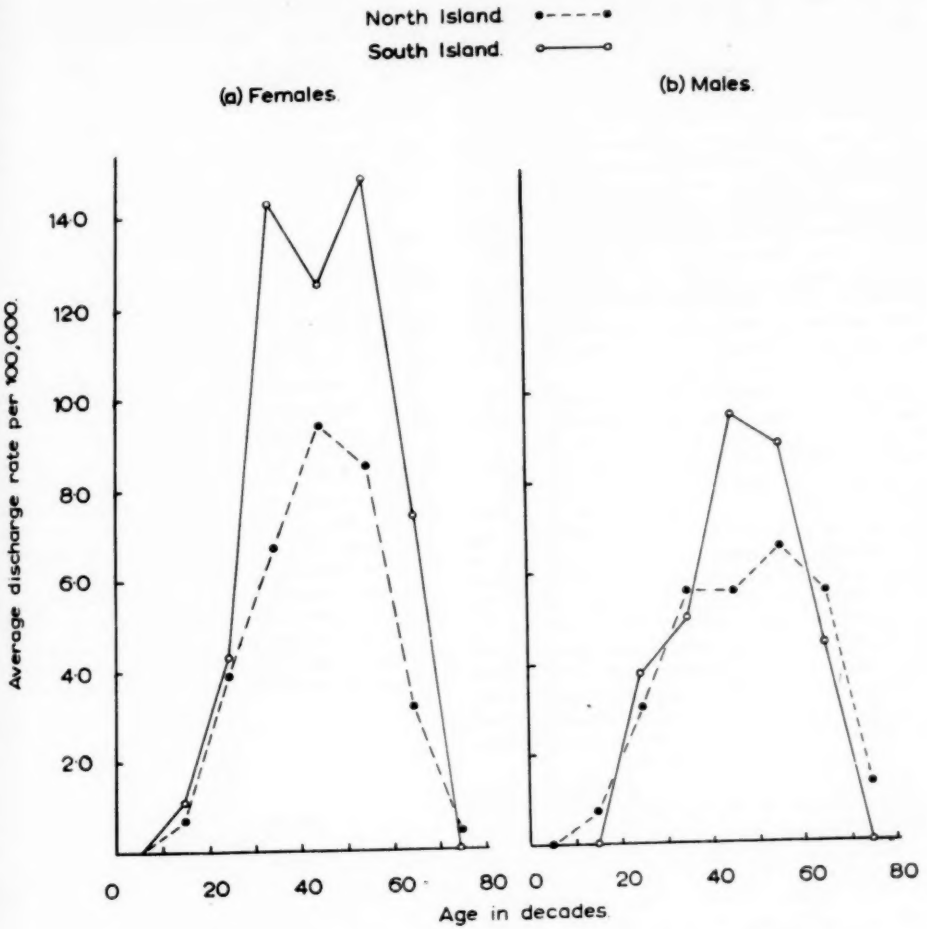

FIG. 3.—Discharge rates (multiple sclerosis) from the New Zealand Public Hospitals (1950–7), by age, sex, and Island of residence.

from South to North in North America. Subsequent field studies have confirmed his conclusion in such measure that it has been suggested (Sutherland, 1956, 1959) that an index of local prevalence can be obtained by multiplying the local death rate by the average life expectancy (20 years) (Pratt, Compston, and McAlpine, 1951). Such apparently perilous calculations come close to the observed prevalence (± 20 per cent.) for Louisiana and South Carolina (Kurland, 1952), Ontario (White and Wheelan, 1958), North Scotland (Sutherland, 1956), and Northern Ireland (Allison and Millar, 1954), but not for Minnesota (MacLean, Berkson, Woltman, and Schionneman, 1950), Colorado (Kurland and Dodge, 1953), California (Kurland and Newman, 1953), Montana (Seidler, Nicholl, and Kurland, 1958), or

Nova Scotia (Alter and others, 1960), when the crude mortality rates for State or Country are compared with the best available data on local prevalence.

As we have seen, the fluctuation of the frequency of multiple sclerosis with latitude is now beyond doubt within the United States, and factors which vary with latitude have recently been under scrutiny as possible aetiological influences in the disease (Limburg, 1950; Acheson and others, 1960; Barlow, 1960). This increase in frequency in Northern America holds true in spite of the bias introduced by rising standards of medical care from South to North within that country. Whether or not (as mortality figures suggest) the disease is indeed commoner in the British Isles than in the Northern States will only be settled when compact communities like Rochester, Minnesota, and Kingston, Ontario, are surveyed in Great Britain.

The question, whether the decreasing frequency with which multiple sclerosis is reported as a cause of death in the southern hemisphere as the equator is approached is a real phenomenon or is due simply to a consistent increase in under-reporting, cannot be answered finally at the present time. The slight tendency for motorneurone disease* mortality and the mortality attributed to "senility" also to be related to latitude suggests that the standards of death certification may vary with latitude within the southern hemisphere, although the correlations are not significant. On the other hand, the morbidity data from New Zealand support the mortality data in suggesting that multiple sclerosis is more prevalent in the South than in the North Island. Dean (1949) also confirmed that the disease was extremely rare in South Africans of European stock.

It is also of interest that, when the death rates from multiple sclerosis in the southern hemisphere were correlated with average annual hours of sunshine the relationship was marginally closer than that with latitude, although the difference is not significant. In United States veterans (Acheson and others, 1950) the relationship of winter sunshine at birthplace with frequency of multiple sclerosis was slightly closer than the relationship of latitude.

In conclusion, it may be said that, although no epidemiological question about a chronic disease can be settled by mortality data alone, there is sufficient evidence of an environmental influence to warrant careful field studies in Australia and New Zealand. In particular it is important to know whether, as Sutherland and Eadie (1959) have suggested, Brisbane, in spite of its position on the edge of the tropical zone, really has an appreciable prevalence of multiple sclerosis, unlike Natal and Louisiana in similar situations. If this is so, the study of such an exception might be rewarding. In South America, Chile and Argentina, which extend over 30° of latitude, deserve study. It would also be of interest to know the prevalence of multiple sclerosis in the large German community in Santa Catarina State in Brazil (27·5°S.).

*This disease was selected because it also requires a high standard of diagnosis and because its probable genetic aetiology suggests that it may be evenly distributed except in isolated communities.

SUMMARY

A study has been made of the mortality ascribed to multiple (disseminated) sclerosis in the white settler populations of Australia, New Zealand, and South Africa within the period 1950–9. A close relationship has been found between the multiple sclerosis death rate (age-adjusted) and latitude, which is very similar to the relationship which exists for the death rate within the English-speaking countries in the northern hemisphere. Some comparisons are made with the mortality rates ascribed to amyotrophic lateral sclerosis during the same period. These fluctuate much less widely. It is concluded that while no final answer can be obtained from mortality data alone, the distribution of deaths ascribed to multiple sclerosis in the Southern hemisphere is sufficiently suggestive of an environmental factor (or factors) similar to those operating in the northern hemisphere to justify local prevalence studies.

Hospital discharge rates for multiple sclerosis for the two islands of New Zealand are also given.

REFERENCES

Acheson, E. D. (1959). *J. chron. Dis.*, **10,** 469, 481.
—— and Bachrach, C. A. (1960). *Amer. J. Hyg.*, **72,** 88.
——, ——, and Wright, F. M. (1960). *Acta psychiat. scand.*, **35,** Suppl. 147, 132.
Allison, R. S., and Millar, J. H. D. (1954). *Ulster med. J.*, **23,** Suppl. 2.
Alter, M., Allison, R. S., Talbert, O. R., and Kurland, L. T. (1960). *Wld. Neurol.*, **1,** 55.
Annual Abstract of Statistics (1958). No. 95, "Sunshine Averages, 1921–50".
Bammer, H., and Schaltenbrand, G. (1960). *Acta psychiat. scand.*, **35,** Suppl. 147, p. 57.
Barlow, J. S. (1960). *Ibid.*, **35,** Suppl. 147, p. 108.
Brooks, C. E. P. (1950). "Climate in Everyday Life". Benn, London.
Dassel, H. (1960). *Acta psychiat. scand.*, **35,** Suppl. 147, p. 64.
Dean, G. (1949). *Brit. med. J.*, **1,** 842.
Department of Health, Education and Welfare. "Annual Report, National Office of Vital Statistics". Washington, D.C.

Garnier, B. J. (1958). "The Climate of New Zealand". Arnold, London.
Georgi, F., and Hall, P. (1960). *Acta psychiat. scand.*, **35,** Suppl. 147, p. 75.
Gorczynski, W. (1948). *C. R. Soc. Sci. Lettres*, Varsovie Classe III, Année 1947.
Hyllested, K. "Disseminated Sclerosis in Denmark". (1956). Jorgensen, Copenhagen. (*Cited by* Alter and others, 1960).
Kurland, L. T. (1952). *Amer. J. Hyg.*, **55,** 457.
—— (1958). *J. chron. Dis.*, **8,** 378.
—— (1959). "Pathogenesis and Treatment of Parkinsonism". Thomas, Springfield, Ill.
—— and Dodge, H. J. (1953). *Neurology*, **3,** 577.
—— and Moriyama, I. M. (1951). *J. Amer. med. Ass.*, **145,** 725.
—— Mulder, D. W., and Westlund, K. B. (1955). *New Engl. J. Med.*, **252,** 649, 697.
—— and Newman, H. W. (1953). *Calif. Med.*, **79,** 381.
Limburg, C. C. (1950). *Res. Publ. Ass. nerv. ment. Dis.*, **28,** 15.
MacLean, A. R., Berkson, J., Woltman, H. W., and Schionneman, L. (1950). *Ibid.*, **28,** 25.
Miller, H., Ridley, A., and Schapira, K. (1960). *Brit. med. J.*, **2,** 343.
Pratt, R. T. C., Compston, N. D., and McAlpine, D. (1951). *Brain*, **74,** 191.
Siedler, H. D., Nicholl, W., and Kurland, L. T. (1958). *Journal-Lancet*, **78,** 358.
Sutherland, J. M. (1956). *Brain*, **79**, 635.
—— and Eadie, M. J. (1959). *Brit. med. J.*, **2,** 824.
Weather Bureau, Department of Transport, Pretoria. "Sunshine and Cloudiness in South Africa".
White, D. N., and Wheelan, L. *Quoted by* Kurland (1958).
W.H.O. (1948). "Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death (6th Revision)", W.H.O., Geneva.
—— (1956). Epidemiological and Vital Statistics Report", vol. 9, p. 595.

Brit. J. prev. soc. Med. (1961), **15**, 126

INCIDENCE OF GASTRIC AND DUODENAL ULCER IN THE ROYAL NAVY IN 1955

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In a previous paper (Baron and Vaughan Jones, 1958) we discussed the difficulties of estimating the prevalence of gastric and duodenal ulcer. We concluded, as had Doll (1952), that a population survey was the method of choice, and we studied the whole British Army of 1955. We later studied the Royal Air Force of 1955 (Baron and Vaughan Jones, 1960) and we present here the data on the third armed service. In 1955 there were 122,903 men (officers and other ranks) in the Royal Navy including the Royal Marines. We have analysed the morbidity from peptic ulcer diagnosed that year for the first time.

Methods

In 1955, diagnoses of gastric duodenal or peptic ulceration were made in 377 naval personnel. There were errors of coding in 28 cases, but the remaining 349 documents (92·6 per cent.) were examined. A total of 76 was excluded as the initial diagnosis had been made before 1955.

The remaining 273 men were admitted to hospital in 1955 for the first time with a diagnosis of peptic ulcer. In 36 patients the diagnosis was presumptive, in nine because they had not had a barium meal, and in 27 because the results were negative. The remaining 237 patients were considered to have had proven ulcers according to the criteria used in the previous papers:

(1) 186 duodenal and seventeen gastric ulcers were shown by barium meal to the satisfaction of the radiologist concerned. Three patients (1·3 per cent.) had both gastric and duodenal ulcers.
(2) Nineteen duodenal and eight gastric ulcers were found at laparotomy to have perforated.
(3) Two duodenal ulcers were found at laparotomy for haematemesis.
(4) Five duodenal and two gastric ulcers were found at elective gastric surgery.
(5) Neither gastroscopy nor autopsy contributed to this series.

In some cases the diagnosis was established by more than one method in the course of the year, so that the total of the above groups (245) is more than the number of patients (237) considered to have proven ulcers.

The three patients with ulcers in both the stomach and the duodenum are omitted from further consideration. We have analysed the remaining 234 patients by age at diagnosis, by frequency of presentation with perforation, and by attack rates as calculated from average figures for officers and men of the Royal Navy in 1955.

Results

Table I shows the number of ulcers diagnosed, the ratio of duodenal to gastric ulcers, and the attack rates per 1,000 men at different ages. The number and proportions of ulcers which presented by perforation are shown in Table II.

Incidence of Duodenal Ulcer

The overall incidence of newly-diagnosed duodenal ulcers was 1·7 per 1,000 men. The attack rate rose to a maximum in the 35 to 39-year age group. The overall incidence and distribution were similar to that in the British Army and Royal Air Force, with the peak incidence less than one-tenth of the civilian ulcer incidence.

Duodenal/Gastric Ratio

Table I shows that the ratio of duodenal to gastric ulcers in all age groups was 8 : 1. This figure is similar to that in the British Army in 1955 (7·65 : 1) and lower than that in the Royal Air Force (12·4:1).

The ratio under 45 years of age in the present series is 8·9 : 1, similar to the British Army (8·2 : 1) and lower than the Royal Air Force (12·7 : 1).

TABLE I

NUMBER OF CASES, DUODENAL/GASTRIC RATIO, AND ATTACK RATES BY AGE

Age (yrs)	Number of Men	No. of Ulcers Diagnosed		Ratio Duodenal : Gastric	Annual Incidence per 1,000 Men		
		Duodenal	Gastric		Duodenal Ulcer	Gastric Ulcer	Either
15–19	26,131	21	2	10·5 : 1	0·8	0·08	0·9
20–24	49,819	98	15	6·5 : 1	2·0	0·30	2·3
25–29	20,885	47	1	47·0 : 1	2·3	0·05	2·3
30–34	9,670	13	1	13·0 : 1	1·3	0·10	1·4
35–39	8,557	20	3	6·7 : 1	2·3	0·35	2·7
40–44	4,625	5	1	5·0 : 1	1·1	0·21	1·3
45+	3,216	4	3	1·3 : 1	1·2	0·93	2·2
All Ages	122,903	208	26	8·0 : 1	1·7	0·21	1·9

TABLE II

NUMBER AND PERCENTAGE OF ULCERS WHICH PRESENTED BY PERFORATION, BY AGE

Age (yrs)	Duodenal			Gastric		
	Total Diagnosed	Perforated		Total Diagnosed	Perforated	
		Number	Per Cent.		Number	Per Cent.
15–19	21	1	5	2	2	100
20–24	98	6	6	15	4	27
25–29	47	7	15	1	0	—
30–34	13	1	8	1	0	—
35–39	20	3	15	3	0	—
40–44	5	0	—	1	1	100
45+	4	1	25	3	1	33
All Ages	208	19	9	26	8	31

PERFORATED ULCERS

Table II shows that 9 per cent. of duodenal ulcers and 31 per cent. of gastric ulcers presented with perforation, figures similar to those in the Army and Royal Air Force.

NAVY/ARMY/AIR FORCE DIFFERENCES

There are no major differences in the incidence of peptic ulcer between the Army and the Navy. The Royal Air Force has a significantly lower incidence of gastric ulcer, and therefore a higher duodenal/gastric ratio than either of the other two services. It was suggested in our previous article (Baron and Vaughan Jones, 1960) that this difference might be related to social and educational differences in the composition of the Army and Air Force. It is therefore most interesting that the incidence of gastric ulcer in the Royal Navy resembles that in the British Army rather than that in the Royal Air Force.

SUMMARY

All in-patient case summaries of peptic, gastric, and duodenal ulcer patients in the Royal Navy in 1955 were sought, and 92·6 per cent. were examined and assessed. In 1955, 208 duodenal and 22 gastric ulcers were diagnosed for the first time in approximately 123,000 male personnel of the Royal Navy. The overall incidence rates of duodenal and gastric ulcers were 1·7 and 0·2 per 1,000 men respectively. Attack rates were estimated in the various age groups for proven gastric and duodenal ulceration, together with the proportions presenting by perforation. The results are in general similar to the other services, but the incidence of gastric ulcer is the same as in the Army, and double that in the Royal Air Force.

We are indebted to the Medical Director-General of the Royal Navy for making available to us the relevant medical records, and for his permission to publish.

REFERENCES

Baron, J. H., and Jones, R. Vaughan (1958). *Brit. J. prev. soc. Med.*, **12**, 71.

—— —— (1960). *Ibid.*, **14**, 44.

Doll, R. (1952). *In* "Modern Trends in Gastro-enterology", ed. F. Avery Jones, p. 361. Butterworth, London.

Brit. J. prev. soc. Med. (1961), **15**, 128

PREVALENCE OF THE COMMONER EAR, NOSE, AND THROAT CONDITIONS IN THE ADULT RURAL POPULATION OF GREAT BRITAIN

A STUDY BY DIRECT EXAMINATION OF TWO RANDOM SAMPLES

BY

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We have as yet no data on the prevalence, in the general adult population, of diseases of the ear, nose, and throat that are based upon the direct examination of random samples. This study was designed to remedy this defect.

Method

Two random samples of adults, stratified for age and sex, were each drawn from a rural population. We endeavoured to examine as many as possible of the people in these two samples. One of the samples was of the inhabitants of the Vale of Glamorgan in South Wales, and the other was of the inhabitants of Mid-Annandale (Dumfriesshire) in South-West Scotland. The first sample was examined in Glamorgan in July, September, and October, 1957, and the second sample was examined in Dumfriesshire in May and June, 1958. A mobile unit was used for the examinations and this has been described previously (Hinchcliffe and Littler, 1958).

Survey Areas

The survey area in the Vale of Glamorgan comprised 24 parishes centred in Cowbridge, an old market town with a population just over 1,000.

The mid-Annandale area comprised eight parishes which cover an area of 136 square miles and are centred in Lockerbie, a small market town. This is also an agricultural area.

Survey Populations

The populations of both areas had already been defined by private census for unrelated surveys. For our surveys, a random sample of 400 was drawn from each of two populations. Each sample was stratified for age and sex in the manner shown in Table I. We wished to compare the absolute threshold of hearing of our young adults directly with that of another sample. Consequently, we arranged for the age limits and size of our youngest sample to be as stated in Table I.

Table I

STRATIFICATION OF SAMPLE DRAWN IN EACH SURVEY AREA

Age (yrs) ..		18–24	25–34	35–44	45–54	55–64	65–74	All Ages
Sex	Male ..	50	30	30	30	30	30	200
	Female	50	30	30	30	30	30	200

Procedure

For each of the two surveys, individuals in the sample populations were interviewed by home visitors (two in each survey), who explained the investigation and arranged a time for the examination. Individuals were examined in an unselected order.

Investigation of each individual comprised the following, and in this particular sequence:

I. Completion of a questionnaire.

II. A clinical otolaryngological examination

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III. Clinical quantitative gustometry.
IV. A bacteriological examination. (For nasal carriage of *Staphylococcus aureus*)
V. A radiological examination. (For the condition of the paranasal sinuses)
} Welsh Survey Only
VI. Audiometry.

A questionnaire relating to ear, nose, and throat symptoms and audiologic hazards was completed for each individual on his arrival at the mobile unit.

The clinical otolaryngological examination comprised anterior rhinoscopy, mirror examination of the post-nasal space and the larynx, and examination of the ears using a forehead mirror. Tuning-fork tests of hearing were also performed. In the Scottish survey, the clinical examination was restricted to the ears.

If cerumen was preventing adequate visualization of the tympanic membrane, it was removed. For cases of incomplete obstruction, the wax was removed in all but one case by means of a Jobson Horne curette. In the remaining case, one of soft wax, the wax was removed by syringing. For cases of complete occlusion of the external auditory meatus with wax, the following procedure was adopted if instrumental removal with a curette failed:

(*i*) Syringe

if this failed

(*ii*) Olive oil drops put in the meatus three times daily for one week

then

(*iii*) Re-syringe.

If this procedure failed, the condition was denoted "impacted wax". Olive oil was used to soften the wax because, of the commoner cerumenolytics, this was the only one that had been shown to be significantly better than nothing (Hinchcliffe, 1955).

The threshold of perception for taste was determined, using a method of quantitative gustometry (Hinchcliffe, 1958).

The bacteriological examination was directed towards determining the prevalence of the nasal carriage of coagulase-positive *Staphylococcus aureus*. Both nasal vestibules of an individual were sampled with a saline-moistened nylon swab. In the evening, all swabs taken during the day were transported to the laboratory* and stored overnight in the refrigerator. All strains of coagulase-positive staphylococci isolated on culture were typed by bacteriophage tests and tested for resistance to antibiotics.

The radiological examination consisted of taking two occipito-mental views of the skull with a 65 Kv. Newton Victor Model F Portable *X*-ray Unit. This unit was fitted with a special cone to prevent excessive radiation and scatter reaching the patient. The films were independently read by four doctors. The correct interpretation of the radiological appearances for each subject was taken as the modal value of the reports by the four observers. On occasion, a casting vote was required by the author. A group of observers was necessary because of the large inter-observer variation in the interpretation of *x* rays of the paranasal sinuses (Hinchcliffe, 1961). When four observers are used to read these films, the modal opinion for this random sample does not appreciably change with the number of observers employed.

As the final part of the full examination of the individual, the threshold of hearing by earphone listening was measured in each ear at the frequencies 125, 250, 500, 1,000, 2,000, 3,000, 4,000, 6,000, and 8,000 c/s. The additional frequency of 12,000 c/s was used in the Scottish survey. An instrument calibrated to the British Standard Specification for pure tone audiometers was used.

Note: Specimens of the questionnaire completed for each individual, the proforma for reporting the results of the clinical examination, and the proforma for the *x*-ray report may be obtained from the author.

Observers

In the Welsh survey, the questionnaire was completed by the doctor who performed the clinical examination, but, in the Scottish survey, the questions were asked by a non-medically qualified personal assistant. Errors due to inter-observer variation were consequently eliminated within each of the two surveys.

All the clinical examinations in both surveys were performed by one and the same observer.

The audiometric tests in the Welsh survey were conducted by an experienced audiometric technician. In the Scottish survey, all the audiometry was done by the observer who also performed the clinical examinations, and who had had considerable experience with audiometric methods.

Results and Remarks

To avoid separation of the data and the discussion relating to the prevalence of a given condition by

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results and remarks on unrelated conditions, we found it more convenient to combine "Results" and "Remarks" under a number of sub-headings.

We were successful in examining 90 per cent. of the Welsh sample and 95 per cent. of the Scottish sample.

I. Ears

(*a*) *Hearing.*—We have already reported the results on the distribution of thresholds of hearing (Hinchcliffe, 1959b). The absolute threshold of hearing of our normal young adults was shown to be that of the British Standard of the Normal Threshold of Hearing for Pure Tones by Earphone Listening (Hinchcliffe, 1959a).

(*b*) *Acoustic Trauma.*—Table II indicates the prevalence of hazards due to acoustic trauma in the rural male population. The Table indicates the difficulty, even in a rural population, of finding male adults who have not been exposed to acoustic trauma. Whilst 94 per cent. of the total female sample had never fired a gun, only 13·5 per cent. of the total male sample had never fired a gun.

We have already shown that a history of having fired a 0·303 calibre gun accounts for the sex difference in the threshold of hearing at 4,000 c/s for these young adults (Hinchcliffe, 1959a). Further analysis shows that there is a significant ($P < 0·05$) threshold shift at 2,000 c/s, and at this frequency only, in the clinically normal left ears of the males in the youngest age group who had fired a 12-bore rifle only. This was in comparison with the clinically normal left ears of similarly aged males who had never fired a gun. Moreover, there was a correlation between the threshold shift in the left ear and the stated number of rounds of 12-bore ammunition fired. The product moment correlation coefficient was 0·72, which was again significant at the 5 per cent. level. Therefore, the threshold shift in a person's audiogram at 2,000 c/s is, *inter alia*, directly related to the number of rounds of 12-bore ammunition that he has fired in his lifetime. Inspection of the data showed that a more linear relationship was obtained by plotting the threshold shift against the logarithm of the stated number of rounds of 12-bore ammunition fired. Using this relationship, a linear regression equation was derived as follows:

$$y = 3·93 \log x + 1·5 \text{ dB},$$

where x = stated number of 12-bore rounds fired and y = hearing loss at 2,000 c/s in the left ear.

It is surprising that the 12-bore rifle should first show its damaging effects at 2,000 c/s, instead of at 4,000 c/s which is the commonest locus for acoustic trauma. The threshold shift is, however, not marked. A history of having fired a thousand rounds of 12-bore ammunition would, according to the regression equation, produce a shift of less than 15 dB at 2,000 c/s. It is possible that the subsequent development of a traumatic notch due to firing many more 12-bore rounds might show a migration of the locus of maximum threshold shift to 4,000 c/s.

In our youngest sample, two males had used a 0·22 rifle only. Their thresholds of hearing were normal. However, neither had fired more than thirty rounds of ammunition.

(*c*) *Occupational Noise Hazard.*—Table III (opposite) illustrates the prevalence of a current occupational noise hazard,* even in a rural population.

* A potential occupational noise hazard was considered to have existed if the subjects had had, for at least one year, a job where, at one time or another, it was so noisy that they had to raise their voice to be heard.

TABLE II

PREVALENCE OF POTENTIAL ACOUSTIC TRAUMA FROM RIFLE FIRE

Factors in Potential Trauma	Sample	Age Group (yrs)					
		18–24	25–34	35–44	45–54	55–64	65–74
(*a*) Percentage of rural male population who had ever fired a 0·303 calibre rifle	Welsh	33	59	75	55	54	41
	Scottish	49	66	80	72	59	48
(*b*) Median stated number of rounds of 0·303 ammunition fired by those who had used this calibre gun	Welsh	50	200	300	1000	600	650
	Scottish	100	500	200	200	1000	1000
(*c*) Percentage of rural male population who had ever fired a 12-bore rifle	Welsh	53	76	71	69	75	67
	Scottish	31	34	23	28	41	52
(*d*) Median stated number of rounds of 12-bore ammunition fired by those who had used this calibre gun	Welsh	125	150	400	500	1000	500
	Scottish	175	85	550	200	200	300

TABLE III

PERCENTAGE OF RURAL POPULATION WITH A POTENTIALLY NOISE-HAZARDOUS OCCUPATION

Sex	Sample	Age Group (yrs)					
		18–24	25–34	35–44	45–54	55–64	65–74
Male	Welsh	43	48	53	34	46	18
	Scottish	51	58	50	51	44	22
Female	Welsh	15	4	4	0	0	0
	Scottish	29	6	10	10	0	0

These were the individuals who stated that they had a job where, at one time or another, it was so noisy that they had to raise their voice to be heard. Sataloff's criterion for a potentially hazardous noisy environment (Sataloff, 1957).

Table III indicates that occupational noise hazards might be a factor influencing the hearing of rural females also. However, the threshold of the clinically normal youngest group of Scottish women was not significantly different from the British Standard for the Normal Threshold of Hearing. A slight but significant difference in thresholds of hearing was, however, detected between those young women who reported an occupational noise hazard and those who did not.

(*d*) *Conductive Deafness.*—As a qualitative test for conductive deafness, we used the Rinne tuning-fork test. We have shown that this is a valid, reliable test for conductive deafness and, on these particular samples, indicates a conductive deafness of 17 dB or more at 500 c/s (Hinchcliffe and Littler, 1961).

Analysis of the data showed that the prevalence of conductive deafness was significantly lower in the 18 to 24-year age group than in either the 35 to 44 or the 55 to 64-year age groups.

Table IV shows the composition of the conductive deafness in this series. Further analysis attempted to show which of the listed conditions might be responsible for the increase in conductive deafness with age. A one-tailed, exact test for 2×2 Tables showed that there was a significantly ($P < 0{\cdot}02$) greater number of perforations in the 55 to 64-year age group than in the 18 to 24-year age group. There was, however, no significant increase in the prevalence of scarred tympanic membranes with age. There was also a significantly ($P < 0{\cdot}05$) greater number of cases of otosclerosis in the 55 to 64-year age group than in the 18 to 24-year age group. The increase in conductive deafness with age is therefore due to an increase in the prevalence of both chronic middle-ear infections and of otosclerosis.

Our finding that chronic otitis media is the commonest cause of conductive deafness in the general population agrees with the finding of Bentzen and Jelnes (1955). This is in contrast to other samples where otosclerosis may be the commonest cause of conductive deafness (Shambaugh, Wallner, Greene, and Shambaugh, 1933; Kodicek and Garrad, 1954).

TABLE IV

CAUSES OF CONDUCTIVE DEAFNESS IN THE TWO RANDOM SAMPLES OF THE RURAL POPULATION

Condition	Number of Cases	Percentage
1. Chronic suppurative otitis media (Active 7 cases; Inactive 14 cases)	21	32
2. Secondary middle ear sclerosis (*i.e.* scarred tympanic membranes)	20	31
3. Otosclerosis	12	18
4. Radical mastoid operation	3	5
5. Congenital abnormalities	3	5
6. Exudative otitis media	2	3
7. Acquired meatal atresia	1 each	6
8. Chronic adhesive otitis		
9. Impacted wax*		
10. Traumatic conductive deafness (Ossicular chain disruption)		
All Conditions	65	100

* *i.e.* cerumen which was occluding the external auditory meatus and which had resisted the prescribed course of syringing and application of the cerumenolytic.

N.B. It is possible that some of the cases of conductive deafness that were attributed to secondary middle ear sclerosis were, in fact, cases of otosclerosis with scarred tympanic membranes. If we assume that the probability of the occurrence of otosclerosis is unaffected whether the membranes are scarred or not, then the proportion of cases of conductive deafness due to otosclerosis would be raised to about 20 per cent. by introducing this correction. The error is therefore not appreciable.

Extrapolation of our data would indicate a prevalence of conductive deafness in schoolchildren of the order of twenty to thirty cases per 1,000. Falbe-Hansen (1954), in data on Copenhagen schoolchildren, which he says agrees with similar investigations in other countries, gives the value as about 90 cases per 1,000. If, as he says, his data is representative of schoolchildren in general, it indicates that about three-quarters of the cases of conductive deafness in children are not permanent. It is noteworthy that Falbe-Hansen was able to cure three-quarters of his series of conductive deafness by adenoidectomy, radiotherapy, Politzerization, and/or vibration massage.

(*e*) *Presbycusis.*—The prevalence of presbycusis was assessed as follows. We arbitrarily adopted the criterion that a 25 dB speech hearing loss is the level at which a hearing defect commences to handicap an individual.* To convert our data on the ability of individuals to hear pure tones to their ability to hear speech, we used the simple method of transformation described by Fletcher (1950). This gives the

* Steinberg, Montgomery, and Gardner (1940) have stated that a hearing loss of 25 dB at frequencies up to 1,760 c/s begins to be a handicap.

speech hearing loss in a particular ear as equivalent to the average of the two least threshold shifts at the three frequencies 500, 1,000, and 2,000 c/s. Since presbycusis entails a greater deterioration of the hearing at 2,000 c/s than at the other two frequencies, this transformation procedure resolves itself into averaging the threshold shifts at the two frequencies 500 and 1,000 c/s. Table V shows the percentage of individuals in each age group who had clinically normal ears but a computed speech hearing loss of more than 25 dB.

TABLE V

PREVALENCE OF PRESBYCUSIS AS A FUNCTION OF AGE

Age Group (yrs)	Per cent. with Speech Hearing Loss Greater than 25 dB
18–24	0
25–34	0
35–44	0
45–54	4·9
55–64	6·2
65–74	21·0

Percentage of individuals, in each age group, who have a hearing loss for speech (=speech reception threshold) in excess of 25 dB. Speech hearing losses computed from pure tone threshold audiograms.

(*f*) *Tinnitus.*—Table VI shows the prevalence of tinnitus (history of) as a function of age.

There was no correlation between those individuals who experienced, or had experienced, tinnitus and those individuals who worked, or had worked, for more than one year in conditions where it was stated to be so noisy that they had to raise their voice to be heard.

TABLE VI

PREVALENCE OF TINNITUS AS A FUNCTION OF AGE

Age Group (yrs)	History of Tinnitus
18–24	21
25–34	27
35–44	24
45–54	27
55–64	39
65–74	37

Percentage of individuals in each age group who, at one time or another, have noticed noises in their ears or their head.

(*g*) *Vertigo.*—At one time or another, 23 per cent. of the sample had experienced vertigo. There was a tendency for the proportion of people who had experienced, or did experience, vertigo to increase with age. Table VII shows the prevalence of vertigo (past or present) as a function of age. Most of the episodes of vertigo that had been experienced were transient and not troublesome, but 15 per cent. of those who had experienced vertigo had had recurrent sustained vertigo with or without other symptoms over a period of at least one year. In the entire sample, two persons gave a classical history of post-traumatic positional vertigo, and one a history indicative of having had vestibular neuronitis. From the history, one case of vertigo appeared to be due to epilepsy, and one case was associated with epilepsy.

TABLE VII

PREVALENCE OF HISTORY OF VERTIGO AS A FUNCTION OF AGE

Age Group (yrs)	History of Vertigo
18–24	17
25–34	20
35–44	19
45–54	23
55–64	35
65–74	29

Percentage of subjects in each age group who suffer, or have suffered, from dizziness or giddiness.

A history of recurrent sustained vertigo occurred in over 3 per cent. of the population and is probably a measure of the prevalence of endolymphatic hydrops, ranging from the mildest to more severe types. One case, however, had been associated with episodic concurrent aphasia, and another with episodic concurrent diplopia. McKenzie (1951) says that symptoms alone are a poor foundation for a diagnosis, and Ménière's disease is no exception. We have therefore given a more restricted connotation to this condition by including the objective sign of a low tone threshold shift in the criteria for its acceptance in this study (see Table IX for computed prevalence).

In view of the demonstrated positive correlation between the prevalence of arteriosclerosis and that of vertigo (Droller and Pemberton, 1953), the bulk of the vertiginous histories (especially the episodes of transient vertigo) in the older individuals may well be arteriosclerotic in origin. It would, however, be difficult to attribute this etiology to vertigo experienced by younger individuals.

(*h*) *Wax.*—Cerumen completely occluded 12·5 per cent. of the external auditory meatuses in the total sample. There was a significant sex difference. 23 per cent. of males had one or both meatuses occluded as compared with 13 per cent. of females. Table VIII shows how the clearance of the cerumen in these cases was achieved.

TABLE VIII

METHODS OF CLEARANCE OF CERUMEN FROM COMPLETELY OCCLUDED MEATUSES

Method of Clearance		
	Wax curette	50
	Syringing only	33
	Syringing plus drops	12
Ears remaining Occluded		5
Total		100

(*i*) *Ear Disease.*—Table IX shows the measured prevalence of the principal aural conditions, together with the limits within which the true prevalence probably lies.

TABLE IX

PREVALENCE OF THE COMMONER AURAL CONDITIONS IN THE RURAL POPULATION

Condition		Measured Prevalence (per 1,000)	95 per cent. Confidence Limits of Prevalence
Scarred tympanic membranes		128	105–154
Perforated tympanic membranes		30	19–45
Chalk patches		28	18–43
Chronic otitis externa	Primary	18	9–30
	Secondary	4	1–12
Inactive chronic suppurative otitis media		18	9–30
Active chronic suppurative otitis media		11	5–21
Otosclerosis		11	5–21
Ménière's disease		11	5–21
Cortical mastoid operations		8	3–18
Exudative otitis media		5	1–14
Radical mastoid operations		4	1–12
Chronic adhesive otitis		3	0·3–10

II. NOSE

(*a*) *Clinical and Radiological Findings.*—Here the greater portion of the data is provided by the radiological and bacteriological investigations. The prevalences of the principal nose and sinus conditions are shown in Table X.

TABLE X

PREVALENCE OF THE COMMONER NASAL CONDITIONS IN THE RURAL POPULATION

Condition		Measured Prevalence (per 1,000)	95 per cent. Confidence Limits of Prevalence
Acute rhinitis		11*	3–28
Nasal polyposis		17	6–36
Chronic suppurative maxillary sinusitis	Criterion *i*	25	12–48
	Criterion *ii*	17	6–36
Maxillary sinusitis (radiographic index)		107	77–144
Frontal sinus agenesis	Bilateral	39	22–65
	Unilateral	104	75–141
	Either	143	109–184

* If we include the Scottish sample and restrict the criteria to those who answer in the affirmative to the question "Do you have a cold now?", the prevalence is 25 per 1,000 population.

Several inter-relationships were investigated. There was no significant correlation between those individuals who had upper respiratory symptoms in general, or nasal signs in general, and those who had radiological changes in the maxillary sinuses. There was also no demonstrable association between individuals with nasal symptoms or clinical signs and those with absent frontal sinuses. The following significant ($P < 0{\cdot}05$) positive correlations were, however, found.

(*i*) Between those individuals who had upper respiratory symptoms and those who had positive rhinoscopic signs (χ^2 with Yates' correction = 4·4).

(*ii*) Between those individuals with a history of mucopurulent nasal discharge and those with radiological signs (generalized opacity, fluid level, mucosal thickening, or cyst) in one or both antra (χ^2 with Yates' correction = 4·46). The probability of radiological signs in respect of the maxillary sinuses was 0·19 if there was no history of mucopurulent discharge, but 0·44 if there was a history.

(*iii*) Between those individuals with visible evidence of mucopus in the nose and those who were nasal carriers of coagulase-positive *Staphylococcus aureus* (χ^2 with Yates' correction = 3·98). The probability of being a nasal carrier was increased by 58 per cent. when mucopus was present in the nose.

Apart from the presence of pus or mucopus in the nose, the only other abnormality on rhinoscopic examination which is worthy of mention is the allergic type nasal polyp. The measured prevalence of nasal polyposis was seventeen cases per 1,000. This affords some objective measure of the prevalence of nasal allergy.

No sex differences were demonstrable in the clinical or radiological data. Cysts of the antra appeared to be commoner in younger adults.

The absence of any correlation between individuals with upper respiratory symptoms in general and those with radiological changes in the maxillary sinuses is almost certainly due to the varied aetiology responsible for upper respiratory symptoms in general. Moreover, many individuals with chronic sinusitis are symptom free. If we become more specific and consider a single symptom only, and one (*e.g.* mucopurulent nasal discharge) that might be considered to be related, on logical analysis, to

radiological changes in the sinuses (on the assumption that these are principally infective in their aetiology) then we do find a significant correlation between symptoms and radiological signs.

The precise importance that can be attached to particular upper respiratory symptoms in these surveys is uncertain. There is considerable inter-observer variation in reported prevalences of respiratory symptoms in general (Cochrane, Chapman, and Oldham, 1951; Schilling, Hughes, and Dingwall-Fordyce, 1955). Analysis of our results showed that, in the Scottish sample, nasal block was stated to be, or to have been, experienced by a significantly greater number of women in the 45 to 54-year age group than in the 65 to 74-year age group. In view of the similar ecological conditions obtaining for the two groups, this is unlikely to be the case. Moreover, in the case of males in the Scottish sample also, there is a significant difference between these two age groups for those who stated that they did suffer, or had suffered, from nasal block. But in this case a higher prevalence is recorded in the older age group. There is therefore some doubt about the validity of the question referring to the symptom of nasal block.

In view of the uncertain validity of upper respiratory symptoms in epidemiological studies, we sought to concentrate on radiological methods to assess the prevalence of sinusitis. In analogy with the electrocardiographic index of the prevalence of ischaemic heart disease described by Thomas, Cochrane, and Higgins (1958), we suggest a radiological index of the prevalence of sinusitis, as shown in Table X. It should be stressed that this is an index of sinusitis that may be neither chronic nor suppurative. In children, the radiological changes in the sinuses may be permanent in only about one half (Bjuggren, Kraepelièn, and Lind, 1952) or one quarter (Nitsch, 1957) of the cases. For our sample of adults, however, we consider that it is unlikely that temporary sinus conditions account for such an appreciable portion. In view of the statement of van Dishoeck and Franssen (1957) that an allergic factor is demonstrable in more than 60 per cent. of cases of chronic purulent sinusitis, the suspicion may arise that, even if the radiological changes are permanent, they may not have an infective aetiology. This statement, however, applies to the aetiology and pathogenesis, and not to the pathology, of the condition. A consideration of the work of Buch (1949) indicates that our radiological index of maxillary sinusitis is, allowing for a margin of temporary changes, a measure of the prevalence of chronic suppurative sinusitis, irrespective of the possibility of an allergic factor in its pathogenesis. Moreover, by using a group of observers to read the *x*-rays of the sinuses, it is also possible that we have reduced, if not eliminated, the false positive results (10 per cent.) that Buch obtained.

In an attempt to eliminate the factor of temporary change in our radiological measurement of the prevalence of sinusitis, we can combine our clinical data with our radiographic data. Where the prevalence of chronic suppurative sinusitis is shown in Table X, Criterion *i* (see Appendix) permits the use of data from both the questionnaire and the clinical examination, but Criterion *ii* utilizes the results of the questionnaire only. These alternatives have been used because criteria for the occurrence of chronic suppurative sinusitis have not yet been defined for epidemiological purposes. It is possible that future surveys may not be conducted by, or be able to make use of, an observer experienced in clinical otolaryngological examinations. Moreover, Criterion *ii* is more rigid in its definition, but because it is more restricted, and in view of the remarks made by Daggett (1954), this criterion will give a deflated value of the prevalence of active chronic suppurative maxillary sinusitis. The symptom of purulent or mucopurulent nasal discharge was chosen to establish Criterion *ii* because it is the predominant symptom in chronic infections of the paranasal sinuses (Thornell, 1944). Moreover, we have shown that it is the only symptom that is significantly correlated with radiological changes in the sinuses.

A consideration of our results, together with Buch's data on the proportion of active cases of undiagnosed sinusitis, would indicate that the prevalence of active chronic suppurative maxillary sinusitis in the rural population of Great Britain is of the order of 50 per 1,000 population. In view of the statement by Meer, Andrassy, Bodo, Codrea, Foldessy, and Sirban (1957) that the prevalence of sinusitis in agricultural workers is low, our measured prevalence value is probably exceeded in urban areas. Individuals with positive findings in respect to the sinuses should be followed up to investigate the persistence of both radiological and clinical changes.

This appraisal of the prevalence of sinusitis illustrates very well the necessity for rigid criteria in defining a given condition.

Our measured prevalence of 4 per cent. for bilateral frontal agenesis compares well with values of 3 and 5 per cent. reported for European races by Chalmagne (1953) and by Longo and Blandino (1956) respectively.

(*b*) *Bacteriology: Nasal Carriage of* Staphylococcus Aureus.—The prevalence of the nasal carriage of both penicillin - sensitive and penicillin - resistant strains of coagulase-positive *Staphylococcus aureus* is shown, as a function of age and sex, in Table XI. Fig. 1 shows the prevalence of the nasal carriage of the staphylococcus (sensitive or resistant strains) as a function of age, with sex as parameter.

TABLE XI

PREVALENCE OF NASAL CARRIAGE OF *STAPHYLOCOCCUS AUREUS*, BY AGE GROUP AND SEX

Sex	Cases	Age Group (yrs)					
		18–24	25–34	35–44	45–54	55–64	65–74
Female	Number examined	46	28	23	27	26	23
	Penicillin sensitive strains	14	11	5	10	7	5
	Partially resistant strains*	4	2	1	1	4	3
	Very resistant strains†	3	0	2	0	0	0
	No growth	25	15	15	16	15	15
Male	Number examined	49	28	28	29	24	26
	Penicillin sensitive strains	19	8	9	6	4	4
	Partially resistant strains*	4	1	1	2	0	0
	Very resistant strains†	1	2	1	0	1	0
	No growth	25	17	17	21	19	22

* Circle of inhibition about one half of the diameter shown by the Oxford staphylococcus.
† No noticeable inhibition of growth around penicillin containing disc.

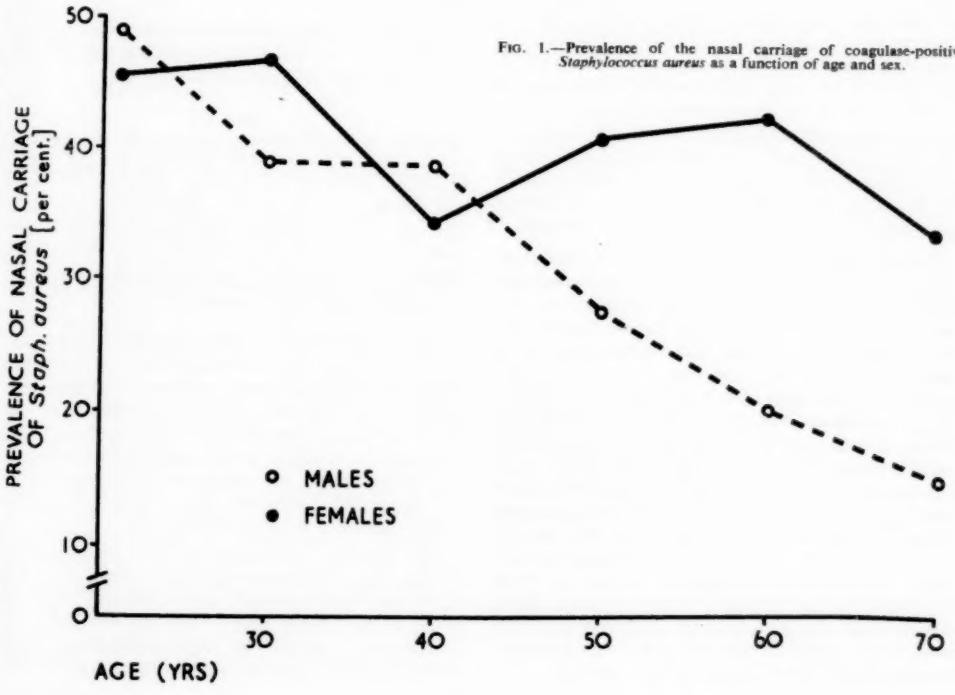

FIG. 1.—Prevalence of the nasal carriage of coagulase-positive *Staphylococcus aureus* as a function of age and sex.

The graph shows that the prevalence of the nasal carriage of coagulase-positive *Staph. aureus* declines linearly with age in males. Unpublished data by Williams (1947) also indicate that there is a decline in the prevalence of the nasal carriage of this organism with age. Our extrapolated predicted value of 53 per cent. prevalence at the age of 15 years is identical with the prevalence value reported by Williams (1947) for the second decade. Presumably, there is a reversal of the age *v.* prevalence trend in this age period since the data given by Cunliffe (1949) show that nasal carriage increases from about 20 per cent. at the age of 1 year to about 60 per cent. at the age of 9 years. Hallman (1937) found the peak prevalence for the nasal carriage of *Staph. aureus* in the age group 13 to 19 years (60·5 per cent.). As Cunliffe (1949) says, it is tempting to speculate on these variations in the nasal flora. The gradual fall with age may be a reflection of an increasing immunity, but may, on the other hand, result from a less intimate contact with the community than occurs during the school years.

A significant sex difference can be demonstrated in our data when the two oldest age groups are combined. Analysis of the data of Elwood (1951) for the nasal carriage of staphylococci by 126 patients on first attendance at hospital shows a sex difference (female greater than male) which is significant at the 1 per cent. level. It is possible that the divergence of the female trend line from the male trend line with increasing age reflects reinforcement of the female reservoir by the greater staphylococcal reservoir in children. However, Brzezinski (1939) showed that the male population has a higher antistaphylolysin titre than the female population.

It is to be noted that Miles, Williams, and Clayton-Cooper (1944) have demonstrated seasonal differences in the nasal carriage of *Staph. aureus*, the peak prevalence being found in the months of July and October. These were also the months during which we conducted this particular survey. Our prevalence rates may therefore represent maximal values. Moreover, recent data given by McDonald, Miller, Jevons, and Williams (1960), for the prevalence of the nasal carriage of staphylococci by Royal Air Force recruits, indicate a somewhat lower prevalence than we obtained for our corresponding group of males. A consideration of the data of Miles and others (1944) and of McDonald and others (1960) for the prevalence in males for rural areas and for the appropriate geographical region suggests that our prevalence data should be multiplied by a factor of 0·7 to give a representative average value devoid of seasonal fluctuations.

The higher prevalence of nasal carriage of *Staph. aureus* in individuals with visible evidence of mucopus in the nose was not associated with any particular phage type. The association was probably due to the increased moisture in the nasal cavity (Thomson, 1960).

III. Mouth and Pharynx

(*a*) *Teeth.*—The edentulous state as a function of age is shown in Fig. 2 (opposite). For comparison, data obtained on a group of coal-miners by Cochrane (1960) is also shown in the Figure.

There is a linear rise in the edentulous state with age. Moreover, there is a significant shift to the left of the curve for Cochrane's sample of English coal-miners. This indicates that miners reach a state of dental insufficiency about 5 years earlier than men living in a rural community. The two lines probably express the prevalence of the edentulous state of the country as a whole.

(*b*) *Taste.*—The distribution of the taste threshold has already been reported (Hinchcliffe, 1958). In the sample where taste was tested, there was one case of taste blindness.

(*c*) *Tonsils.*—Nearly twice as many people had had their tonsils removed in the Scottish sample as in the Welsh sample (see Table XII).

TABLE XII

PREVALENCE OF TONSILLECTOMIZED INDIVIDUALS (PER CENT.) BY AGE GROUP*

Sample	Age Group (yrs)					
	18–24	25–35	35–44	45–54	55–64	65–74
Welsh	24	16	19	0	8	4
Scottish	40	26	24	22	15	9

* Calculated from answers to questionnaire.

This difference was significant at the 0·1 per cent. level, *i.e.* highly significant. Of those who had had the operation, 4 per cent. had had it on two occasions. A definite age at which tonsillectomy was performed was given by 91 per cent. of those in the sample who admitted to having had the operation. The distribution of the stated age when tonsillectomy was performed is shown in Fig. 3; 14 per cent. of the stated ages were in excess of 20 years.

The regional difference in the prevalence of tonsillectomy is interesting. In a survey of 1,434 Royal Air Force recruits born between 1930 and 1936, Carne (1956) found that the prevalence of tonsillectomy was slightly, but significantly, greater in recruits from Scotland than in those from Wales.

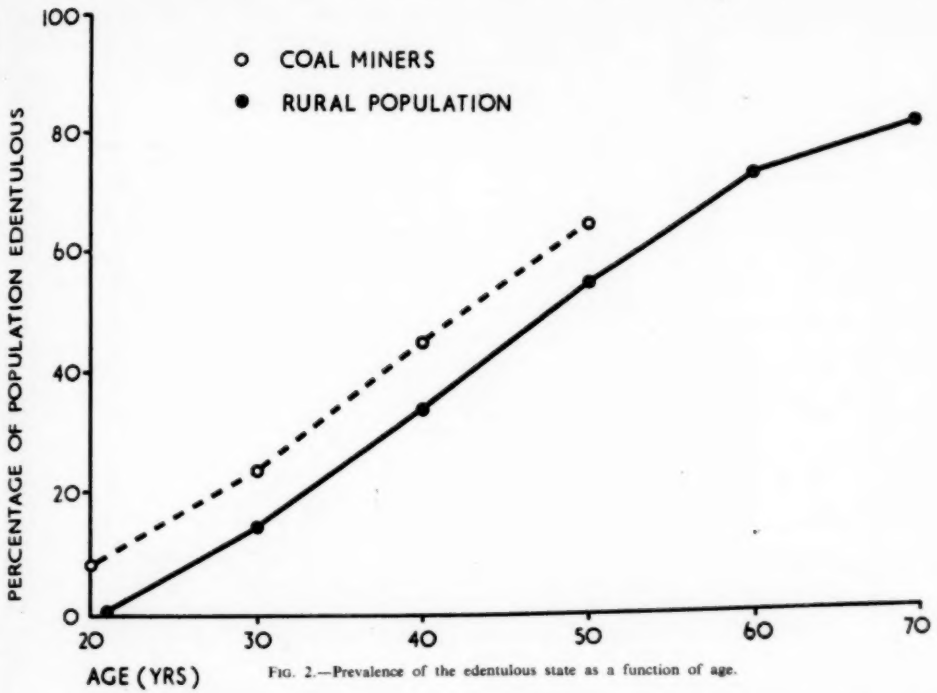

FIG. 2.—Prevalence of the edentulous state as a function of age.

However, an almost identical proportion (just over 30 per cent.) of recruits from rural areas had had the operation in both Scotland and Wales. Carne's finding that 32 per cent. of his entire sample had had tonsillectomy is identical with our value for our comparable age group 18 to 24 years) from the Scottish and Welsh sample combined. It is therefore probable that the combined data on tonsillectomy from the author's surveys are representative of the country as a whole. As we have shown, however, there are more localized regional differences. To what extent are these discrete regional differences due to differences in the attitude of the medical profession to this operation? There appeared to be no regional differences in the prevalence of disease of the ears, nose, or throat. Moreover, Table XII shows a decline in the prevalence of tonsillectomized individuals with age, but there was no associated trend in the prevalence of ear, nose, and throat diseases with age, and *there was no significant difference in the prevalence of ear, nose, and throat disease in general, or of radiological sinus signs in particular, amongst those who had had the operation and those who had not.* A consideration of the data shown in Table XII and Fig. 3 (overleaf) indicates that the prevalence of tonsillectomy in Great Britain was on the increase at least until about the year 1941.

Our finding that the most common age for the operation was 5 years (see Fig. 3) agrees with that of Rolleston (1939). Carne found that the most common age at which the operation had been performed in his sample was 7 years. Our data suggest that this discrepancy may be due to a tendency that is now developing to delay the age for the operation. Carne states that it is reasonable to assume that the peak prevalence for the operation is associated with the physiological hypertrophy of the tonsils which occurs from the fourth to the eighth years. In this respect, it is worth noting the recent demonstration of Okabe (1958) that tonsillar hypertrophy in school children showed no correlation with either physique or intelligence.

IV. Post-Nasal Space and Larynx

In the Welsh sample, where mirror examination of the post-nasal space and larynx was attempted,

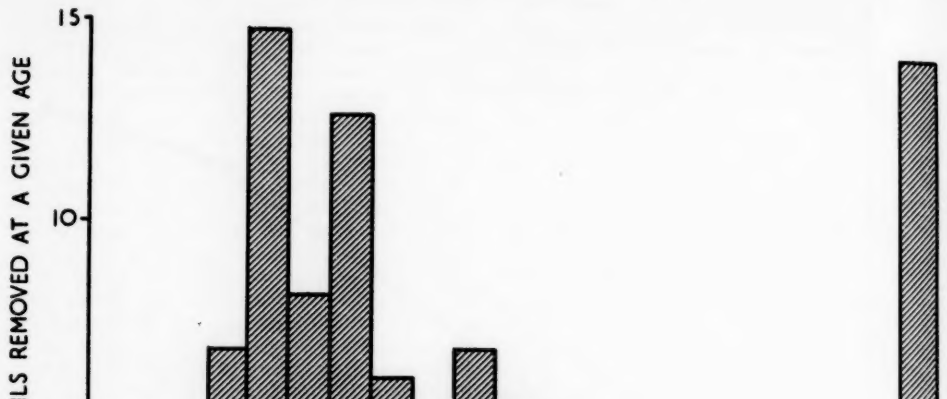

FIG. 3.—Distribution of stated age at which tonsils were removed.

one male and four female subjects refused to have the examination. Moreover, in the others, one or both locations could not be adequately visualized in 30 per cent. of the sample. Topical anaesthetization was not employed. Furthermore, abnormal findings were infrequent in the post-nasal spaces and larynges that were adequately visualized. Consequently, it is recommended that mirror examinations of the post-nasal space and larynx should be discontinued in surveys of this type.

An impression was obtained that laryngeal and post-nasal examinations were frequently more difficult to perform in these predominantly normal populations than in clinic populations. Presumably more co-operation might be expected in people who attend hospital with symptoms referable to the upper respiratory tract than in individuals selected at random from the general population.

GENERAL REMARKS

General agreement, with few exceptions, in the results for the two areas sampled indicates that the composite results for the two main samples are probably representative of the adult rural population of the country as a whole. Moreover, since age differences were more usually not demonstrable for disease prevalence, the ascertained prevalences will be generally applicable. It is possible that our samples were too small to demonstrate age differences.

CONCLUSIONS

The general agreement between the results of the two surveys means that we can now state the broad otorhinolaryngological characteristics of the rural population of Great Britain. Further surveys should investigate urban, specific industrial, and, perhaps, specific geographical populations. The feasibility of

measuring the prevalence of ear, nose, and throat conditions has now been established.

SUMMARY

Two random samples, stratified for age and sex, of the rural population in Great Britain were otologically and audiometrically examined. Good agreement between the two sets of results indicates the general applicability of the calculated prevalence values to the rural population of the country as a whole. One of the samples was also examined radiologically with respect to the paranasal sinuses, and nasal swabs were also taken to ascertain the prevalence of the nasal carriage of *Staphylococcus aureus*. The feasibility of both the radiological and bacteriological examinations for epidemiological purposes was, at the same time, demonstrated.

My thanks are due to Dr. T. S. Littler, Director of the Medical Research Council Wernher Research Unit on Deafness, to Dr. J. C. Gilson, Director of the Medical Research Council Pneumoconiosis Research Unit, and to Dr. C. Clayson, Physician Superintendent, Lochmaben Hospital, for their help and advice. I am also indebted to Prof. A. L. Cochrane and the Medical Research Council Epidemiological Team for their assistance in this project.

REFERENCES

Bentzen, O., and Jelnes, K. (1955). *Acta otolaryng.* (*Stockh.*), **45,** 189.

Bjuggren, G., Kraepelièn, S., and Lind, J. (1952). *Ibid.*, **42,** 287

Brzezinski, S. (1939). *Schweiz. Z. allg. Path. Bakt.*, **2,** 18.

Buch, A. (1949). *Acta otolaryng.* (*Stockh.*), Suppl. 77.

Carne, S. (1956). *Brit. med. J.*, **2,** 19.

Chalmagne, J. (1953). *Acta oto-rhino-laryng. belg.*, **7,** 280.

Cochrane, A. L. (1960). Personal communication.

——, Chapman, P. J., and Oldham, P. D. (1951). *Lancet*, **1,** 1007.

Cunliffe, A. C. (1949). *Ibid.*, **2,** 411.

Daggett, W. I. (1954). *Practitioner*, **173,** 668.

Dishoeck, H. A. E. van, and Franssen, M. G. C. (1957). *Pract. oto-rhino-laryng.*, **19,** 502.

Droller, H., and Pemberton, J. (1953). *J. Laryng.*, **67,** 689.

Elwood, J. S. (1951). *J. Path. Bact.*, **63,** 745.

Falbe-Hansen, J. (1954). *Acta otolaryng.* (*Stockh.*), **44,** 157.

Fletcher, H. (1950). *J. acoust. Soc. Amer.*, **22,** 1.

Hallman, F. A. (1937). *Proc. Soc. exp. Biol.* (*N.Y.*), **36,** 789.

Hinchcliffe, R. (1955). *Brit. med. J.* **2,** 722.

—— (1958). *Acta otolaryng.* (*Stock.*), **49,** 453.

—— (1959a). *Ibid.*, **50,** 411.

—— (1959b). *Acustica* (*Stuttgart*), **9,** 303.

—— (1961). *J. Laryng.*, **75,** 101.

—— and Littler, T. S. (1958). *Ann. occup. Hyg.*, **1,** 114.

—— —— (1961). *J. Laryng.*, **75,** 201.

Kodicek, J., and Garrad, J. (1954). *Ibid.*, **68,** 406.

Longo, G., and Blandino, G. (1956). *Boll. Mal. Orecch.*, **74,** 414.

McDonald, J. C., Miller, D. L., Jevons, M. P., and Williams, R. E. O. (1960). *Proc. roy. Soc. Med.*, **53,** 255.

McKenzie, W. (1951). *J. Laryng.*, **65,** 70.

Meer, P., Andrassy, L., Bodo, A., Codrea, A., Foldessy, Z., and Sirban, I. (1957). *Oto-rino-laring* (*Bucureşti*), **2,** 115.

Miles, A. A., Williams, R. E. O., and Clayton-Cooper, B. (1944). *J. Path. Bact.*, **56,** 513.

Nitsch, K. (1957). *Mschr. Kinderheilk.*, **105,** 251.

Okabe, H. (1958). *Igaku Kenkyu*, **28,** no. 12, p. 10 (4548). (English summary, p. 27).

Rolleston, C. (1939). *Brit. J. Child. Dis.*, **36,** 251.

Sataloff, J. (1957). "Industrial Deafness". McGraw-Hill.

Schilling, R. S. F., Hughes, J. P. W., and Dingwall-Fordyce, J. (1955). *Brit. med. J.*, **1,** 65.

Shambaugh, G. E., Wallner, L. J., Greene, L. D., and Shambaugh, G. E. Jr. (1933). *Arch. Otolaryng.* (*Chicago*), **18,** 430.

Steinberg, J. C., Montgomery, H. C., and Gardner, M. B. (1940). *J. acoust. Soc. Amer.*, **12,** 291.

Thomas, A. J., Cochrane, A. L., and Higgins, I. T. T. (1958). *Lancet*, **2,** 540.

Thomson, S. (1960). Personal communication.

Thornell, W. C. (1944). *Proc. Mayo Clin.*, **19,** 470.

Williams, R. E. O. (1947). *Quoted by* Cunliffe (1949). (Personal communication).

See Appendix Overleaf

APPENDIX

CRITERIA ADOPTED FOR GIVEN ABNORMALITIES WHERE THESE ARE NOT SELF-EVIDENT

Condition/Disease	Criteria
Ageusia	Inability to detect or recognize any of the test solutions for the four taste qualities when in a concentration corresponding to 100 gusts (*i.e.* sucrose 115 gm/100 ml.; sodium chloride 34.7 gm/100 ml.; quinine sulphate 0.0417 gm/100 ml.; tartaric acid 0.794 gm/100 ml.).
Conductive Deafness	A true Rinne negative response.
Conductive Deafness, Congenital	Conductive deafness with an anatomical abnormality of the conducting mechanism of the ear that is identifiable as of the congenital type.
Conductive Deafness, Traumatic	Stationary conductive deafness immediately following a head injury and with intact, unscarred tympanic membrane.
Ménière's Disease	Recurrent, spontaneous, sustained vertigo for a period of not less than one year, and associated with nausea or vomiting but not with loss of consciousness. Imperforate tympanic membranes. Partial/complete sensori-neural hearing loss for low tones in one or both ears.
Otitic Blast Injury	Centrally-perforated tympanic membrane with no evidence of polyp or cholesteatoma and with non-progressive ipsilateral deafness dating from a blast injury.
Otitis, Chronic Adhesive	Conductive deafness in the presence of an opaque, immobile, unscarred tympanic membrane.
Otitis Externa, Chronic Inactive	External auditory meatus shows generalized scaling of walls with no evidence of cerumen.
Otitis Externa, Chronic Active, Primary	Meatus occupied or lined with discharge. Imperforate tympanic membrane. No concurrent or recent aural pain.
Otitis Externa, Chronic Active, Secondary	Meatal walls generally inflamed, with no evidence of wax formation and associated active chronic suppurative otitis media.
Otitis Media, Chronic Suppurative, Inactive	Perforated tympanic membrane with no history of blast injury and no evidence of aural discharge.
Otitis Media, Chronic Suppurative, Active	Perforated tympanic membrane with no history of blast injury but with evidence of aural discharge—either direct visible evidence or observation that cotton-wool on a wire carrier is moistened.
Otitis Media, Exudative	Intact tympanic membrane showing hair-line and/or bubbles.
Otosclerosis	Visibly normal tympanic membrane in presence of true Rinne negative response.
Rhinitis, Acute	Inflamed nasal mucosa together with affirmative answer to the question: "Have you a cold at the moment?"
Sinusitis, Maxillary (Radiographic Index)	Presence of generalized opacity of, mucosal thickening of, or "fluid level" in, one or both maxillary sinuses, as determined by the modal opinion of four readers of the occipto-mental skull films.
Sinusitis, Chronic Suppurative Maxillary — Criterion *i*	As for maxillary sinusitis, together with (*a*) a history (in questionnaire) of one or more symptoms referable to nose for not less than one year; and (*b*) visible evidence of mucopus or pus in nose, or a history of purulent/mucorpurulent discharge for at least one year.
Criterion *ii*	As for maxillary sinusitis, together with a history of mucopurulent discharge for at least one year.

SOCIETY FOR SOCIAL MEDICINE

Proposed Programme of the 5th Annual Meeting, September 28-30, 1961

LONDON SCHOOL OF HYGIENE AND TROPICAL MEDICINE

SYMPOSIUM

The General Practitioner and Preventive Medicine

Chairman: Professor W. S. Walton
Introduction: E. M. Backett
Child Health: R. M. Acheson
Mental Health: S. P. W. Chave
Occupational Health: Lord Taylor

SCIENTIFIC SESSIONS

(1) **Natality:**

ALISON MACDONALD (*Guy's Hospital, London*): Prematurity, associated defects and early post-natal factors.
I. M. LECK (*Birmingham University*): Season of birth in relation to other environmental variables.
VICTORIA COFFEY (*Trinity College, Dublin*): Maternal anaemia and congenital defects.

(2) **Physique: Non-Infectious Disease**

GILLIAN FOWLER (*Guy's Hospital, London:* introduced by R. M. ACHESON): Fat in the Rhondda Fach.
C. R. LOWE (*Birmingham University*): Arterial pressure, physique and occupation.
M. SUSSER (*Manchester University*): Fluctuations in peptic ulcer mortality.
J. A. HEADY, J. N. MORRIS, A. KAGAN, and P. A. B. RAFFLE (*London Hospital and London Transport Executive*): Direct physical measurement and size of uniforms in the study of busmen's physique.

(3) **Geographical Epidemiology**

A. M. MCFARLAN (*Bristol University*): Spot maps of infectious diseases: early and late examples relating to cholera, poliomyelitis, hepatitis, measles and malaria.
P. J. LAWTHER, R. E. WALLER, and MAUREEN HENDERSON (*St. Bartholomew's Hospital, London*): The effects of weather and air pollution on bronchitic patients.

(4) **Industrial Medicine**

J. BOYD (*London School of Hygiene*): Mortality among luminizers.
MURIEL NEWHOUSE (*London School of Hygiene*): Occupational dermatoses in an automobile factory.
A. S. FAIRBAIRN (*London School of Hygiene*): Survey of railway accidents.
D. D. REID (*London School of Hygiene*): Occupational and social factors in miners' mortality.

(5) Mental and Behavioural Disorders

R. W. PARNELL (*Warneford Hospital, Oxford*): Measuring the significance of signs and treatment in psychiatry.

NANCY GOODMAN and J. TIZARD (*Maudsley Hospital, London*): The prevalence of mental defect in the London area with special reference to children.

M. POWER (*London Hospital*), introduced by J. N. MORRIS: A pilot study of juvenile delinquency in East London.

(6) Informal Discussion on

Training and Careers in Social Medicine, opened by I. M. RICHARDSON (*Aberdeen University*).